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THE *JOURNAL OF PERIODONTOLOGY* is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely will make the Journal a fund of extensive information, readily available to students, teachers, researchers, and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and clinical interest which are presented at the annual meetings of that society. Abstracts of important articles on periodontia that appeared during the year of 1940 are continued in this number.

We are grateful to all who have made available the subject matter from which the abstracts and copies of abstracts have been made, including the authors of the original articles, owners of the journals mentioned in the text of the abstract section, and those who have aided in any way in compiling the material comprising this issue.

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". . .It is not the accretion of knowledge which matters so much as what is happening to one's self in the process of gaining the knowledge.

"Study, like every conscious process, involves an interaction between the inner self and the outer world which results in changes in the individual. Changes in mental habits are occurring continuously, no matter what the kind of mental activity or study may be. These changes may lead toward habits of dawdling and inefficiency, or toward skill and efficiency in the control of mental processes.

"Effective study means effective control of one's mind and the development of useful habits of work. It involves the acquiring of much information and knowledge through reading, observation, experimentation, and association with others; the development of many techniques or abilities such as thinking, solving problems, and acquiring skills; and in the envisioning of goals toward which to direct one's efforts. In the larger sense, study is the application of intelligence to the task of understanding and controlling self and the environment. In learning how to study correctly, we are learning to think and to live . . ." Extract from "What Is Study," by Raymond C. Bandi. *Dental Rays* 17:7. Nov., 1941.

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED FORTY-TWO

Oral Manifestations of Vitamin Deficiencies*†

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THE ROLE of vitamins in the treatment of human ailments has been so popularized, in both the scientific and lay press during the past few years, that if you were to believe all that you read, you would be convinced that vitamins could cure almost any disease. Unfortunately this is not so. However, such great strides have been made in recent years, in our knowledge of the vitamins, both as to their structure and their use, that it requires constant study to maintain an accurate and up-to-date understanding of the subject. In spite of this, there are certain facts which have been corroborated by a sufficient number of investigators to be accepted as established. I will discuss chiefly these without attempting to present the tremendous amount of controversial material that is present in the literature at the present time.

As our knowledge of the chemical structures of the vitamins has increased, we have come to recognize certain clinical entities as being due to a lack of these specific substances. Also we have been able to show both experimentally in animals, and clinically in patients, that an adequate intake of the specific vitamin will cause a disappearance of these symptoms. In addition, there are a number of cases that are of the so-called subclinical type where the patient has only a mild or partial deficiency. Their symptoms may be indefinite and often rather vague and some of them may even be classed as psychoneurotic. Careful evaluation and examination, however, will usually enable one to determine the deficient factor. Also it is possible for a patient to have a deficiency of more than one vitamin. This may give rise to rather confusing symptoms or may be misleading because of the failure to obtain the expected response from treatment.

In order that we may have a clearer understanding of their clinical effects and also that we may assimilate the newer knowledge, we should review briefly the present status of vitamins. Originally, vitamins, as you no doubt know, were considered as only five in number, being designated by the first five letters of the alphabet. As our knowledge has increased, some of these have become subdivided so that now we have a much larger number of definitely identified factors. The vitamins that are concerned with diseases of the oral cavity are chiefly the first

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four: Vitamin A; Vitamin B complex which consists of B_1 or thiamin, B_2 or riboflavin, the p-p factor or nicotinic acid; Vitamin C; Vitamin D. There are several other factors in the B complex but they are not of particular interest in this paper dealing with oral lesions. The other chief vitamins are E, or the antisterility vitamin, and K which is concerned with clotting of the blood.

One of the first vitamins to be isolated was vitamin A. It is found in the vegetable kingdom as carotene in such vegetables as rice, palm oil, chestnuts, carrot roots, yellow corn, and in dairy products. It is found also as vitamin A in the livers of certain fishes. Although the chemistry of this vitamin has long been known it was only within the last ten years that it has been synthesized (1).

There are many different organs in the body that show specific pathological changes as a result of vitamin A deficiency. The eyes are the most classical example of this process. Here there is a softening of the cornea known as keratomalacia and associated with this there is a disturbance in the physiology of vision resulting in night blindness. However, these optic changes are a rather late stage in the process and as a result are of little or no diagnostic help so far as other earlier symptoms are concerned.

The specific pathologic process that takes place in any of the organs affected is first an atrophy of the epithelium followed by a reparative proliferation of the basal cells (2). This new product differentiates into a keratinizing epithelium. This keratinizing process is a relatively late phenomenon, the change most often seen being that of atrophy. The oral cavity, pharynx, nose and nasal accessory sinuses are, comparatively speaking, frequently the site of changes. There is first an atrophic change in the mucous membrane which gives it the appearance of being thin and dry. In the later stages this hyperkeratinization takes place with a piling up of the mucous membrane, even with scale-like formations.

All of this has been learned mostly from experimental work on animals, but also from reports of actual cases in human beings. Wolbach and Howe (3), working on rats, were able to demonstrate the above mentioned changes. They were also able to show that diets deficient in Vitamin A had a definite effect on the formation and growth of teeth. They demonstrated an actual wearing down of the incisors which they felt indicated that a complete cessation of growth could occur since, in the rat, these teeth continue to grow throughout life.

Microscopic studies of the teeth and tooth forming organs showed a shrinking and in some instances a complete disappearance of odontoblasts. The enamel forming cells were either shrunken, atrophic, or had been completely replaced by a narrow layer of non-keratinizing epithelium.

Several investigators have reported finding this process of keratinization involving the salivary glands and their respective ducts. Apparently all of the salivary group can be effected, even the parotid. The pathologic change may destroy the function of the gland by the proliferative process completely filling the acini with the keratinized material. Also this same process may involve the ducts of the glands completely obstructing the lumen. Quite obviously, in either case, there is a diminution or complete absence of salivation. This is often complicated by secondary infection, particularly in those cases where there is obstruction of the ducts.

Orten, Burn, and Smith (4) did some experimental work on vitamin A deficiency with special reference to the formation of odontomas. This work was also on rats. They found that the incisors showed the most striking and consistent changes. They found that the teeth showed a loss of the normal orange pigment and the development of opacities. There was also a distortion of the shape of the teeth with eventual exfoliation of the erupted portions. They also reported that those animals that survived long enough developed odontomas and in a few instances even formed supernumerary incisor teeth.

Sweet and his co-workers (5) report an interesting group of cases of vitamin A deficiency among 203 Chinese, a large percentage of whom were adults. No special mention was made of the teeth but he did find keratinizing changes in the pharynx, larynx and trachea. The apparent absence of pathological changes in the teeth may have

been due to the fact that a large percentage of these cases were adults in whom dental activity, i. e. growth, had ceased.

Blackfan and Wolbach (6) reported a series of thirteen deaths in children, due to vitamin A deficiency. Some of these showed similar changes in the mucous membrane of the oral cavity, pharynx and nares. They suggest that an early diagnosis of vitamin A deficiency can be made by the demonstration of keratinized epithelial cells in scrapings from mucous membrane of the cornea, nose, and mouth. Some of the investigators have maintained that vitamin A is an important adjunct in preventing infections anywhere in the body. As yet there does not seem to be sufficient evidence to support this claim.

In reviewing the effects of vitamin A deficiency we find that it is very important in diagnosing diseases of the oral cavity. The chief effect is on the epithelium, producing first an atrophy followed by a keratinizing metaplasia. This may affect the mucous membrane anywhere in the mouth, including the gingivae, and the salivary glands. Also there is a very definite effect on the formation and growth of teeth due to atrophy and metaplasia of the enamel forming organs.

The present concept of the vitamin B complex is that it is made up of several different factors. Each year it seems that some new factor has been isolated and identified. The main factors to be considered in relation to the oral cavity are: B_1 the antineuritic factor known as thiamin; B_2 known as riboflavin, and the p-p factor now identified as nicotinic acid.

Vitamin B_1 or thiamin is concerned chiefly with the prevention of beri-beri, a disease involving mainly the nervous and the cardiovascular systems. I mention thiamin because it is very popular at the present time in the treatment of patients that are run down generally. It produces an improvement in the appetite and seems to increase the general vitality of the individual. Also there are some encouraging reports of its use in the treatment of certain types of deafness. There are no specific oral lesions that can be attributed to thiamin deficiency. It is possible, though not often seen, to have involvement of the nerve supply to structures about the mouth and throat. Haden (7), in one of his tables lists atrophy of the tongue as one of the symptoms of vitamin B_1 deficiency.

Vitamin B_2, or riboflavin, is found chiefly in the green leaves of rapidly growing plants (9), milk and other dairy products. There is a great deal of dispute about the exact physiology and pathology of riboflavin particularly in experimental animals. According to Hogan (8) it has some function in the physiology of cell oxidation. However, some work (10) has shown that there is a syndrome that is definitely attributable to riboflavin deficiency in man. The most characteristic changes that result from this deficiency are found about the mouth and oral cavity. These are chiefly rhagades at the corners of the mouth. This usually begins as reddened mascerated areas which go on to transverse linea fissures. Along with this, the lips have a reddened denuded appearance. This may be associated with a scaly, slightly gray dermatitis about the naso-labial folds and sometimes about the eyes and ears.

This syndrome is similar to the so-called Plummer-Vinson Syndrome which has been described within the last few years. This syndrome almost always occurs in middle-aged women and consists of similar pathologic changes. There is fissuring at the angles of the mouth with masceration of the skin, and the lips may have a reddened, denuded appearance. In addition to this, there are atrophic changes seen in the tongue similar to those seen in the tongue of pernicious anemia. There can be demonstrated both roentgenologically and endoscopically, an organic stenosis of the cervical esophagus. This is usually associated with an anemia of the microcytic, hypochromic type. No one has, as yet, established the definite etiology of this condition. It is almost always associated with malnutrition, but there is some question as to whether this malnutrition is the cause of the syndrome or whether the dysphagia which is a part of the disease syndrome is a cause of the malnutrition. The similarity between this and the syndrome ascribed to riboflavin deficiency suggests it may also be due to a vitamin deficiency.

The third factor of the vitamin B complex is known as the p-p factor or antipellagra vitamin. A deficiency of this factor likewise produces changes in the oral cavity

that are quite characteristic, particularly when they are associated with the other symptoms of pellagra. There has been much discussion in the past as to the exact factor that is responsible for this disease. There have been several isolated cases that produce similar conditions in animals but since these are many and confusing they will not be mentioned. It is a rather universally accepted fact today that nicotinic acid is the important agent in the prevention of pellagra.

As I have said before the oral manifestations of pellagra are rather characteristic. They are chiefly a glossitis and a stomatitis. Early there is a characteristic redness and swelling at the tip and along margins of the tongue. The tongue is sore and there is excess salivation and dysphagia. As the disease progresses the swelling of the tongue increases and the redness may become intense. There may be ulcerations along the margins of the tongue. Not infrequently there is a thick, white exudate over the surface of the tongue from which Vincent's organisms may be recovered. The buccal mucous membrane, the mucocutaneous surface of the lips, the gums and the palate may likewise be affected. There is often a burning sensation of the tongue and pharynx and the breath is fetid.

These oral lesions are associated with the other signs and symptoms of pellagra namely; a dermatitis which has a peculiar characteristic distribution, a diarrhea, and mental changes.

The clinical entry known as scurvy has long been a well recognized disease. It was described as long ago as the Thirteenth Century. However, it was one Joseph Lind, an officer of the British Navy, who, in his *Treatise on Scurvy*, published in 1753, realized that this was a disorder of nutrition rather than an infectious or occupational disease as had been thought. It was through his work that lemon juice was first included in the rations of the seamen. Holt and Frolich (11) were really the beginners in the experimental work on vitamin C, and it was Szent-Gyorgyi, in 1928, who first isolated a compound he termed "hexaronic acid." Through this work and the work of many subsequent investigators, it has been established that scurvy is due to a deficiency of vitamin C, which is now known as ascorbic acid or cevitamic acid.

According to Dalldorf:

the primary morphologic effects of vitamin C deficiency occur in the intracellular substances of certain mesenchymal derivatives. These can best be described in terms of prototype of these structures, loose connective tissues. Under normal conditions the type cell, the fibroblast, lies in an amorphous ground substance within which fibrils (reticulum) are formed which may in turn become gathered into waxy bands of collagen. In this transformation the fibrils seem to become cemented together by a translucent matrix, the formation suggesting a colloid phenomenon, the setting of a gel. It is precisely this phase of the formation of the intracellular material which may be completely controlled by vitamin C.

While this seems to be highly technical in nature, it will show how exact our knowledge of this vitamin is.

Dalldorf (12) goes on to state that

defective intracellular substances are seen in connective tissues, bones, teeth and are believed to occur in blood vessels. An interesting modification of the process is apparent in partial deficiency, wherein a substitute material is formed in the teeth in place of dentine. This has been spoken of as "osteodentine" since it resembles both materials.

Vitamin C is also supposedly closely related with calcium metabolism.

Clinically, the oral picture of scurvy is quite characteristic and remarkably constant, the disease involving chiefly the teeth and the gums. It usually begins insidiously. The gums become reddened and swollen and bleed quite easily. In severe cases, the swelling of the gums may become so marked as to completely obscure the teeth. As the disease progresses, ulcerations may begin to develop on the swollen gums and this may be followed by secondary infection. Gangrene of the gums may even develop. Granulations may form about the teeth and intractable hemorrhage, which is a frequent complication of scurvy, may occur. Needless to say, this is often accompanied by a fetid breath. Of course, lesions of the gingiva occur only in the presence of teeth and are most severe about deformed and broken teeth.

The teeth themselves show typical pathologic changes. There is rarefaction of the alveolar bone with consequent loosening of the teeth. Pathologically, there may be seen separation of the odontoblast layer from the dentin, and development of hemorrhages within the pulp cavity. Fish and Harris (14) demonstrated defects in enamel and cementum as well as in the dentine. These oral

lesions are, of course, accompanied by the other changes in bone, muscle, skin, and other organs.

In children, the oral signs are usually not prominent unless the teeth have erupted, or are about to erupt. It is not uncommon to see the gums of the upper jaw alone affected, more particularly about the upper central incisors. The gums are swollen, purplish in color and bleed quite easily. Around teeth not yet erupted there may form purplish bags.

Vitamin D, the fourth member of the vitamins, is known as the anti-rachitic vitamin. Rickets is, of course, chiefly a disease of the skeletal system and it is only from this standpoint that it has any relation to disease of the oral cavity. According to the generally accepted view, the pathological changes are brought about by a disturbance in the calcium and phosphorus metabolism. A deficiency results in an inability to maintain and stabilize the levels of calcium and inorganic phosphorus in the blood serum and this in turn results in defective calcification of bones.

It may be said in general, that rickets affects teeth in three ways: first, it delays dentition; second, it leads to the development of enamel defects, and third, it predisposes to dental caries. Eliot (15) and her co-workers have reported quite an interesting series of 451 children followed over a period of time. They were able to conclude "that enamel hypoplasia, particularly the type that is characterized by a symmetrical distribution of defects in those teeth which are formed during infancy and early childhood, is frequently associated with rickets." From their material, they also concluded that the more severe the rickets, the more frequently hypoplastic defects develop. Dental caries was found more often in children with a known history of rickets.

Another group of 150 rachitic children was reported (16) in which special notice was paid to dentition. Out of this group 20 per cent had no teeth at the end of a year and 8 per cent had none at fifteen months.

From the foregoing it can be seen that the disturbance in the development of the teeth depends on the stage of dentition. In infancy it delays eruption of the deciduous teeth and later on it causes defects in the development of the permanent teeth. There is a great deal of argument about the effect of vitamin D deficiency on the development of dental caries. However, it seems fairly well accepted now, that it has no effect on caries in normally formed teeth, but that it leads to caries by causing defective development.

Fortunately, in this country, we do not usually see patients with these advanced states of total deficiency, but only the milder forms due to a partial deficiency. Quite naturally these conditions are more difficult to diagnose and because of this, it behooves us to constantly keep these factors in mind since we have, readily available today, concentrated forms of these vitamins to use in overcoming these deficiencies.

In the case of many of the vitamins it is difficult to state the exact amount that should be accepted as an optimum intake because of the lack of satisfactory tests for normal blood levels. However, in most instances we can make a fairly reliable estimate.

Vitamin A intake for an average adult man weighing 70 Kg should be about 3000 international units daily and from 6000 to 8000 units daily for growing children (17). The chief therapeutic source is cod liver oil, halibut oil or viosterol.

In the case of riboflavin, it is estimated that children up to 10 years of age should have at least 400 units daily or 20 units per hundred calories if the diet consists of over 2,000 calories. In adults the amount should be about 20 units per one hundred calories (18). Therapeutically, this can be administered either in the form of brewers' yeast or synthetic riboflavin. The other important factor of the B complex, the p-p factor, offers more difficulties in estimating its optimum intake level since there is still so much discussion about other factors involved in preventing pellagra. Therapeutically, brewers' yeast and nicotinic acid are the most widely accepted agents.

The vitamin C requirements of the human body, as in the cases of some of the other vitamins, are based mainly on estimates. According to Smith (19), the intake of ascorbic acid should be from about 8 mgs. (new born) to 50 mgs. daily for infants, 22 to 100 mgs. for children, and 20 to 100

mgs. for adults. Ascorbic acid is given chiefly in the form of orange juice, but in cases of severe deficiency the synthetic form may be used.

The vitamin D requirement of individuals is even more difficult to evaluate than some of the other vitamins because of the other factors involved. If the patient's diet does not contain a sufficient amount of calcium and phosphorus the beneficial effect of the vitamin is lost. Taking all these factors into account, it has been estimated (20) that for an infant, the requirement is between 300 and 400 units daily. For children between infancy and adolescence, the daily allowance is about the same plus at least 750 cc. of milk to provide an ample intake of calcium and phosphorus. In adolescents and adults, there does not seem to be any doubt as to the continued need of vitamin D but as yet there are no reliable estimates for these periods. Vitamin D is usually prescribed in the form of cod liver oil, viosterol, or halibut oil.

From the foregoing description of the various vitamins, and the diseases resulting from their deficiencies, it can be seen that a knowledge of their effects is of great importance to any one dealing with diseases of the oral cavity. In almost every case when a deficiency of one or more vitamins exists, the specific one involved can be diagnosed from findings in the oro-pharynx. In briefly reviewing these changes, we find that a vitamin A deficiency will show first, atrophic and then, keratinizing changes in the epithelial lining of the mouth and pharynx and its associated structures; vitamin B_1 deficiency may show some atrophic changes in the tongue; vitamin B_2 or riboflavin deficiency will exhibit changes about the angles of the mouth and lips; a deficiency of the p-p factor or nicotinic acid results in swelling and redness of the tongue, which has a characteristic appearance, along with similar changes in the buccal mucosa; deficiency of vitamin C results in swelling of the gums with bleeding and loosening of the teeth; vitamin D deficiency results in delayed-dentition and defective development of the teeth.

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Biology of the Cementum*†

By BERNHARD GOTTLIEB, M. D., *Dallas, Texas*

In order to understand properly the importance of the cementum in the fixation of the tooth, we must reconsider some embryological facts. The dentin of the root is laid down on the epithelial sheath of Hertwig. This epithelial layer separates the dentin from the surrounding connective tissue. If this were the final arrangement, the completed root would be covered by a continuous coat of epithelium and the pulp at the apical foramen would be the only connection with the connective tissue of the body. But fortunately, the epithelial layer becomes separated very soon from the dentin surface. Connective tissue appears between epithelium and dentin and begins to deposit cementum.

Fig. 1, shows a root in formation with Hertwig's epithelial sheath (a). The epithelium covers the yet uncalcified part of the root separating it from the connective tissue. On the parts (b) the epithelium is separated from the tooth surface by connective tissue forming cementum. If the epithelium remains anywhere on the surface of the dentin, no functional ability can be developed in that area. The epithelium continues to separate the tooth surface from the connective tissue.

Fig. 1—Human root in formation. (a) Hertwig's epithelial sheath. The epithelium at the older parts is detached from the dentin surface. Between epithelium and root surface there is connective tissue forming cementum (b).

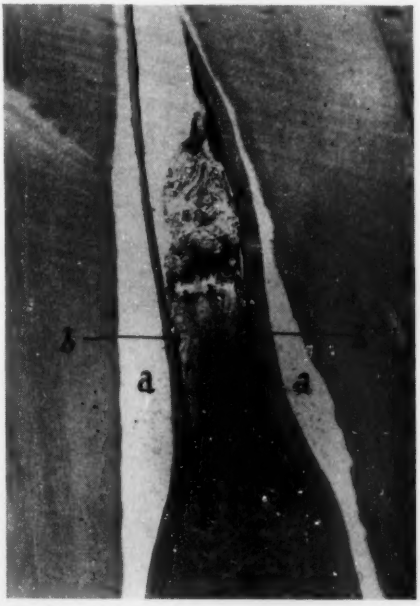

Fig. 2—Two teeth of sheep. (a) Enamel lost by decalcification. (b) Cementum formed on enamel surface. (c) Epithelium attached to cementum.

Formation of cementum on the surface of the enamel increases the functional ability of the tooth. The teeth of herbivora, being of vital importance to these animals, always have cementum on enamel thus making that part of the tooth also functionally useful.

Fig. 2, shows two teeth of a sheep. The enamel, lost by decalcification (a) is covered by cementum (b), the epithelium is attached to the cementum (c). The cementum covering the enamel is used for additional fixation of the tooth.

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†Read before the annual meeting of the American Academy of Periodontology, October 24, 1941.

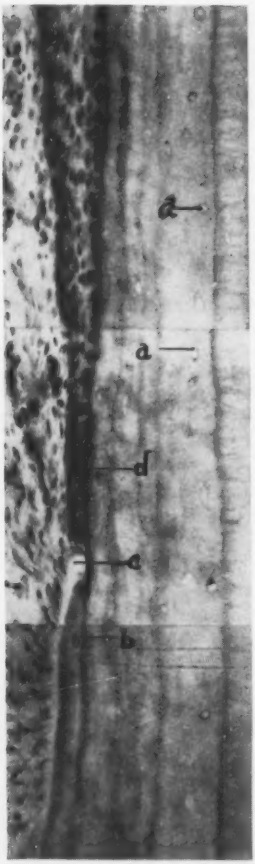

Fig. 3—Epithelial attachment on a pig's tooth. (a) Empty holes. The cells died. (b) A cell in the newly formed cementum. (c) Cementum layer not calcified. (d) Epithelial attachment.

We can also understand more clearly the biology of the cementum by analyzing the situation of the erupted tooth. The tooth, projecting through the soft tissue, makes a hole in the epithelial surface of the body. The epithelial attachment lines that hole. Whenever an opening is made through the *epithelial blanket of the body*, the lining epithelium endeavors to close it by proliferation. We know that the epithelium lining the tooth hole succeeds in doing this after the tooth has been extracted.

The continuous deposition of new cementum layers beneath the epithelial attachment seems to be of great importance. We have the impression that a cementum layer, once formed, undergoes changes which may be described as getting older. We are inclined to believe the vitality of the cementum is decreasing as time goes on, and its ability to work as a barrier against the downgrowth of the epithelium is continually becoming less effective. The epithelium grows over such cementum just as it does over any foreign body which causes a discontinuation of the epithelial cover.

A freshly deposited cementum seems to have the highest vitality and to act as the best barrier. Therefore, the condition is ideal where we find a continuous deposition of new cementum layers. If such a deposition is not made in time, we may be confronted with a downgrowth of the epithelium until the next active barrier of cementum is reached. Fig. 3 shows the epithelial attachment on a pig's tooth. The epithelium stops exactly in front of a new layer of cementum, partly uncalcified. The cementum of pigs' teeth is especially well suited for study of these conditions because it contains cells. These cells enable us to determine the vitality of a particular cementum layer. If we find empty holes without cells, we can look upon such cementum as being dead (a). In the freshly deposited cementum we see a cell in a hole (b).

There is still another angle from which we may see how important it is that new cementum layers be deposited continuously. Suspensory fibers of the periodontal tissues are fixed on the tooth surface by means of the cementum. A newly built cementum layer incorporates into its substance the ends of newly built fibers.

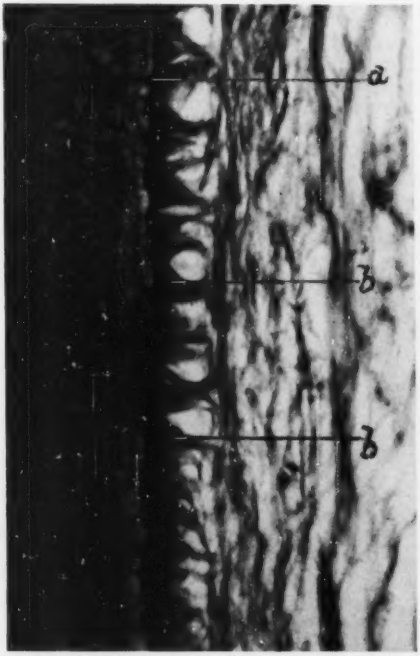

Fig. 4—First formation of cementum embedding periodontal fibers (b). (a) Dentin.

Fig. 5—Older stage than in Fig. 4. (a) Dentin. (b) Cementum entirely calcified. (c) Fibers embedded in the newly forming cementum.

The fixation at the side of the tooth may be disabled in either one of two ways; either by something happening to the fibers or to the cementum. We know that neither cells nor their products, fibers, live as long as the individual lives. Both are continuously exchanged probably in different locations, at varying rates. Newly formed fibers can be attached to the tooth only by newly formed cementum; otherwise they cannot be put into function. Continuous deposition of new cementum layers makes possible the continuous embedding of newly formed fibers. In Fig. 4, we see the first attachment of periodontal fibers to beginning formation of cementum on the dentin surface of the root. Fig. 5, shows a later stage. The periodontal fibers attach again to the surface of the cementum. The existing deeper layers of cementum do not show any traces of fibers; a part of it remains white not taking silver nitrate at all. Fig. 6, shows a further advanced stage. The cementum is already quite thick and the fibers attach again only at the surface. In Fig. 7, we see a thick cementum layer of a herbivora. Here we can trace the periodontal fibers to the middle of the thick-

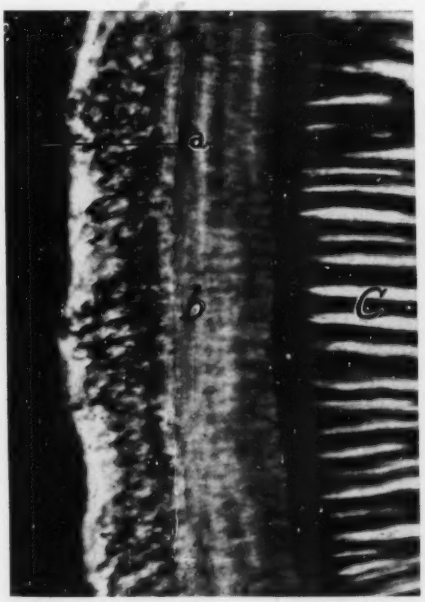

Fig. 6—Older stage than Fig. 5. (a) Dentin. (b) Thick layer of old calcified cementum. (c) Periodontal fibers embedded in the last formed cementum layer.

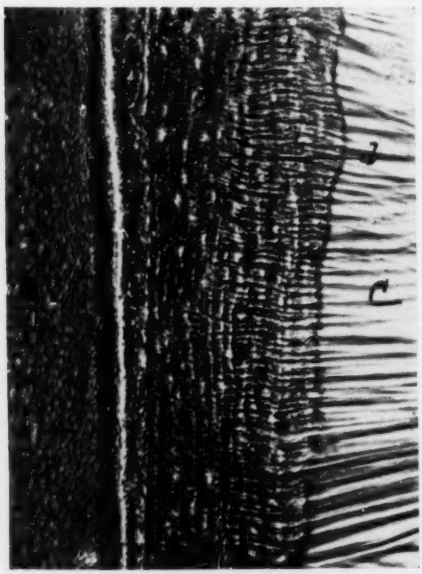

Fig. 7—(a) Dentin. (b) Cementum. (c) Periodontal fibers. The fibers can be traced up to the depth of (d).

ness of the cementum, and we find nature again taking special care of the functional ability of herbivorous teeth. New cementum layers are built before the fibers in the previously built cementum layers are calcified.

It seems that in the cases where we cannot trace the fibers into cementum, those parts of the fibers which are embedded in the cementum calcify too and cannot be distinguished from the calcified cementum. We are inclined to believe that they are fixed only on the last deposited superficial part of the cementum. If such a layer fails to form, the fibers of the periodontal membrane lose their continuity with the calcified parts embedded in the cementum, and functional ability is lost.

In case of resorption of a part of the tooth surface, the fibers lose entirely any connection with the tooth. The function can then only be restored by deposition of cementum on the resorbed surface. The newly deposited cementum can fix the newly formed fibers. Fig. 8, shows tooth resorption with beginning repair. We see new fibers (a) embedded in the newly formed repairing cementum (b).

There is still another reason why it is probably of advantage to have cementum deposited continuously. The resorption of hard tissues seems to depend to some extent upon their quality. It seems probable that noncalcified, newly formed tissue is more resistant to resorption than older calcified tissue. The continuous deposition of new cementum layers means the presence of a coat of noncalcified cementum protecting the surface of the root against resorption. Such a protection may be expected to work under usual conditions. Fig. 9, shows resorption of a deciduous tooth of a sheep. The calcified dentin was resorbed while a long strip of uncalcified dentin (a) resisted resorption. Fig. 10, shows the compressed periodontal membrane of a human tooth (a); near by rear resorption is going on (b). The tooth opposite the resorption remains intact.

In order to maintain the tooth intact as an organ, the enamel of its unerupted parts as well as the cementum must be protected against the possibility of becoming resorbed

Fig. 9—Deciduous tooth of a sheep in resorption. A strip of uncalcified dentin (a) resists resorption, while the calcified dentin has been peeled off by resorption.

Fig. 8—Tooth resorption in repair. New periodontal fibers (a) are embedded in the newly formed repair cementum (b). (c) Dentin.

by the adjacent connective tissue. If the protective measures disappear, or lose their ability to protect, the connective tissue surrounding the tooth may start resorption and apposition as it is accustomed to do with bone throughout life.

Fig. 10—(a) Compressed periodontal membrane. (b) Rear resorption. (c) Intact cementum.

We know of many instances in which the protecting qualities of the cementum surface apparently are lost. The adjacent connective tissue no longer maintains a respectful attitude toward the tooth surface. The result may be beginning resorption with or without ankylosis, or ankylosis without resorption. The quality or degree of injury done to the cementum surface may be decisive.

It seems that the continuous deposition of new cementum layers on the surface of the root is of the same protective ability for the root surface as is the separating and protective value of the epithelium to the enamel before eruption. It protects the tooth against a resorbing attack by the connective tissue. It protects the tooth surface against ankylosis. If the cementum surface is damaged, both can take place.

Nature does the majority of things properly. If she fails to do so in vital organs, they finally disappear, and if the badly cared for organs are of vital importance, a whole species may disappear. Research means to listen and to observe nature's work carefully in order to understand how she does things. If we imitate nature's way correctly and do not drive a wrong road we will have reliable guidance for our prophylactic and therapeutic endeavors.

If these ideas about the biology of the cementum are correct, it is then our task to find out just how nature provides for continuous cementum deposition, and having done so, to imitate the procedure.

In Memoriam

C. S. KRAMER, JULY, 1941

SAUL S. SCOTT, OCTOBER, 1941

The Effect of Deficiencies of the Filtrate Fraction of the Vitamin B Complex and of Nicotinic Acid on Teeth and Oral Structures*†

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INTRODUCTION

Recent writings on the value of the various vitamin B fractions have included a number of clinical observations dealing with the effects of partial vitamin B complex deficiencies on oral structures (2) (3) (8) (10) (20) (21). Until a few years ago, vitamin B was believed to be a single essential food accessory, namely, thiamin or vitamin B_1. It was soon suspected, however, that there existed also one or more other factors whose exact nature was not clearly understood. Then, in rapid succession, riboflavin (B_2) was identified in 1934 by Kuhn (11), pyridoxin (B_6) in 1935 by P. György (9), a "filtrate fraction" in 1935 and 1936 by Elvehjem and Koehn (4) and Lepkovsky, Jukes, and Krause (12) and finally nicotinic acid in 1937 by Elvehjem, *et al* (5) (6) (7). It is now generally recognized that there are still more fractions of the B family, as, for instance, B_3, B_4, B_5, factors W, Y, L and M; however, their identity has not been definitely established as yet. Some of these may be identical with biotin, inositol, paraaminobenzoic acid and other substances recently shown to be vitamins.

Vitamin B deficiency experiments have been conducted during the past five years with rats and dogs in our laboratories (13) (14) (15) (16) (17) (18) (19). This has given us an opportunity to study the conditions of the oral cavities of these animals and to investigate the histologic changes which were produced in dental and paradental tissues by deficiencies of the filtrate fraction and of nicotinic acid.

"Filtrate fraction" as used in this report refers to all members of the vitamin B complex not adsorbed on fuller's earth at approximately pH_5. The adsorbed members are chiefly thiamin, riboflavin and pyridoxin. The filtrate contains pantothenic acid as well as one or more unknowns. Many of the symptoms observed in these filtrate fraction deficient animals are due to lack of pantothenic acid, but not all their symptoms may be ascribed to this deficiency. This has been shown by later experiments in which crystalline pantothenate was fed. It seems best, therefore, for the present to continue the use of the expression "filtrate fraction."

EXPERIMENTAL MATERIAL AND ROENTGENOGRAPHIC ASPECT

From a large series of experiments, and for the following report, six dogs were selected (Table 1), three of which were fed diets complete except for the filtrate fraction of the vitamin B complex (Dogs 10, 12, and 15), while two others received neither the filtrate fraction nor nicotinic acid (Dogs 11 and 13), and the sixth lacked only nicotinic acid (Dog 14). Two additional dogs (1 and 4) of approximately the same age were used as controls and protected against any of these vitamin deficiencies.[1] Duration of the experiments and ages are given in Table 1.

It may be noted in Table 1 that the three dogs in Group I had a more extended period of experimentation and were older at the time of autopsy than those of Group II. This may explain the differences in morphologic changes between the groups, as is shown later. Dogs 10 and 12 were litter mates, black, part Boston Bull female terriers, while No. 11 was an airedale of a much larger build. The others were cocker spaniels.

*From The George Williams Hooper Foundation for Medical Research and the Division of Dental Medicine of the College of Dentistry, University of California, San Francisco, Calif.
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†Read before the Annual Meeting of the American Academy of Periodontology, Houston, Texas, October 24, 1941.
[1]For detailed information on diet composition and behavior of these dogs during the course of the experiment, the reader is referred to publications 14, 15, 16, 17.

Table 1: DISTRIBUTION OF ANIMALS AND EXPERIMENTAL ARRANGEMENT

Group	*Dog No.*	*Sex*	*Duration of Experiment (In Days)*	*Age in Days*		*Type of Deficiency*
				When placed on diet	*At autopsy*	
Control	1	♂	291	39	330	None
	4	♂	281	39	320	None
Group I	10	♀	310	35-42	375	No filtrate fraction
	12	♀	246 deficiency 184 curative D	"	490	No filtrate fraction
	11	♂	343	"	388	No filtrate fraction No nicotinic acid
Group II	15	♂	148	"	188	No filtrate fraction
	14	♀	256	"	296	No nicotinic acid
	13	♂	150	"	184	No filtrate fraction No nicotinic acid (Salt-free for 95 days)

At the time of autopsy, the oral cavities were thoroughly examined for pathologic changes and compared with recordings at the beginning of the experiment. The upper and lower jaws were then dissected and their halves roentgenographed.[1]

For comparison of jaw and tooth development, a half jaw of control dog 4 is reproduced in Fig. 1, while those of the six experimental animals are given in Figs. 2 and 3.

It is interesting to note that the three older animals (Table 1, Group 1) show roentgenographically severe pathologic changes (Fig. 2) which are absent in the controls (dogs 1 and 4). The dental structures of dogs 10 and 12 (Fig. 2, A and B),

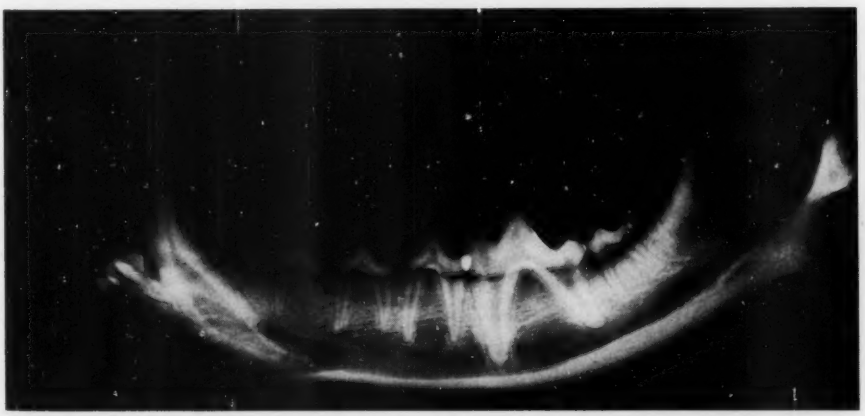


Fig. 1—Roentgenogram of control dog 4. Note even distribution of bone trabeculae throughout the jaw and absence of marginal atrophy. Teeth are in good alignment and have long, straight roots.

[1]Appreciation is expressed to Dr. Gordon M. Fitzgerald of the Dental Roentgenology Department of the University of California for his co-operation and courtesy in making the roentgenograms of all dogs.

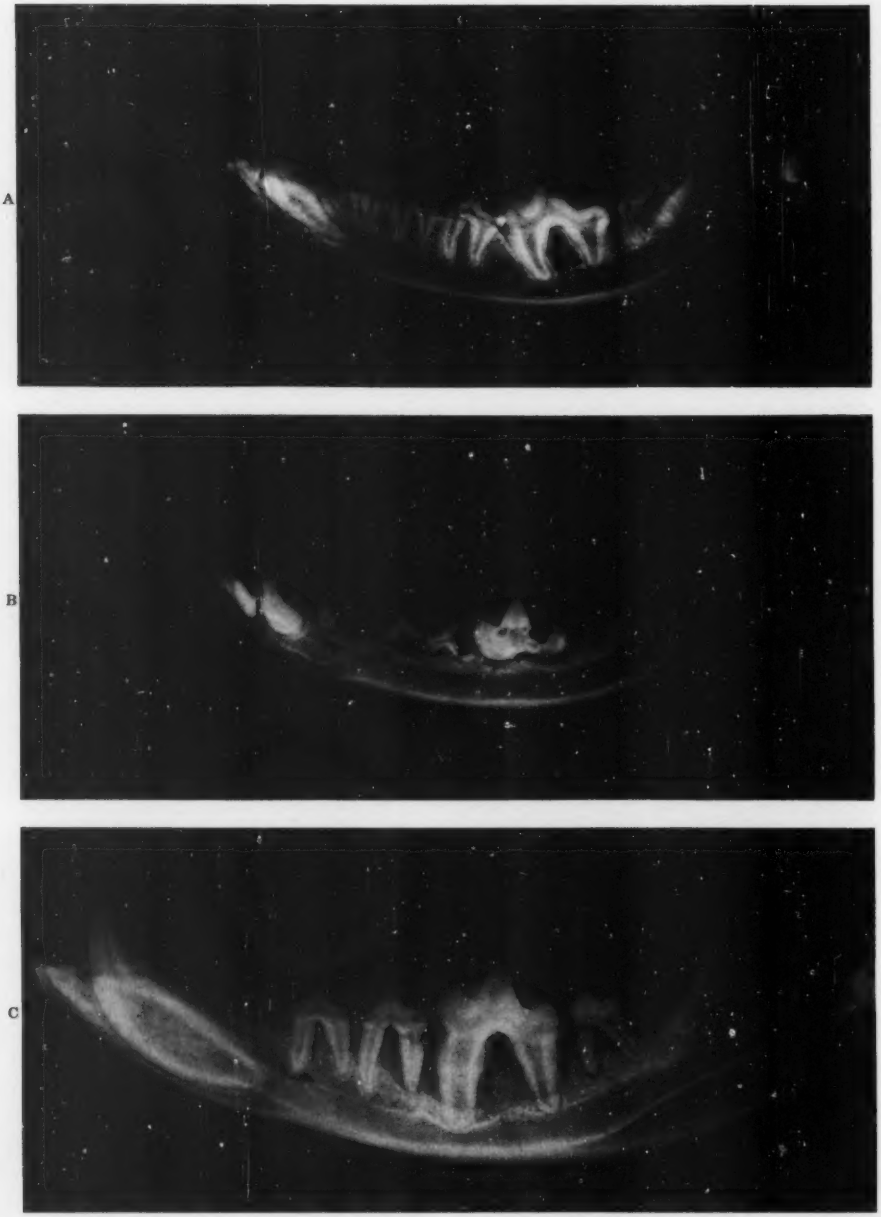


Fig. 2—The jaws of A, B and C are those of dogs 10, 12 and 11, respectively. Note surface caries of tooth crowns and osteoporosis of the jaw in A; severe dental caries and absence of roots in B; inflammation of entire jaw bone structure in C, with marked marginal bone atrophy and paradental pocket formation.

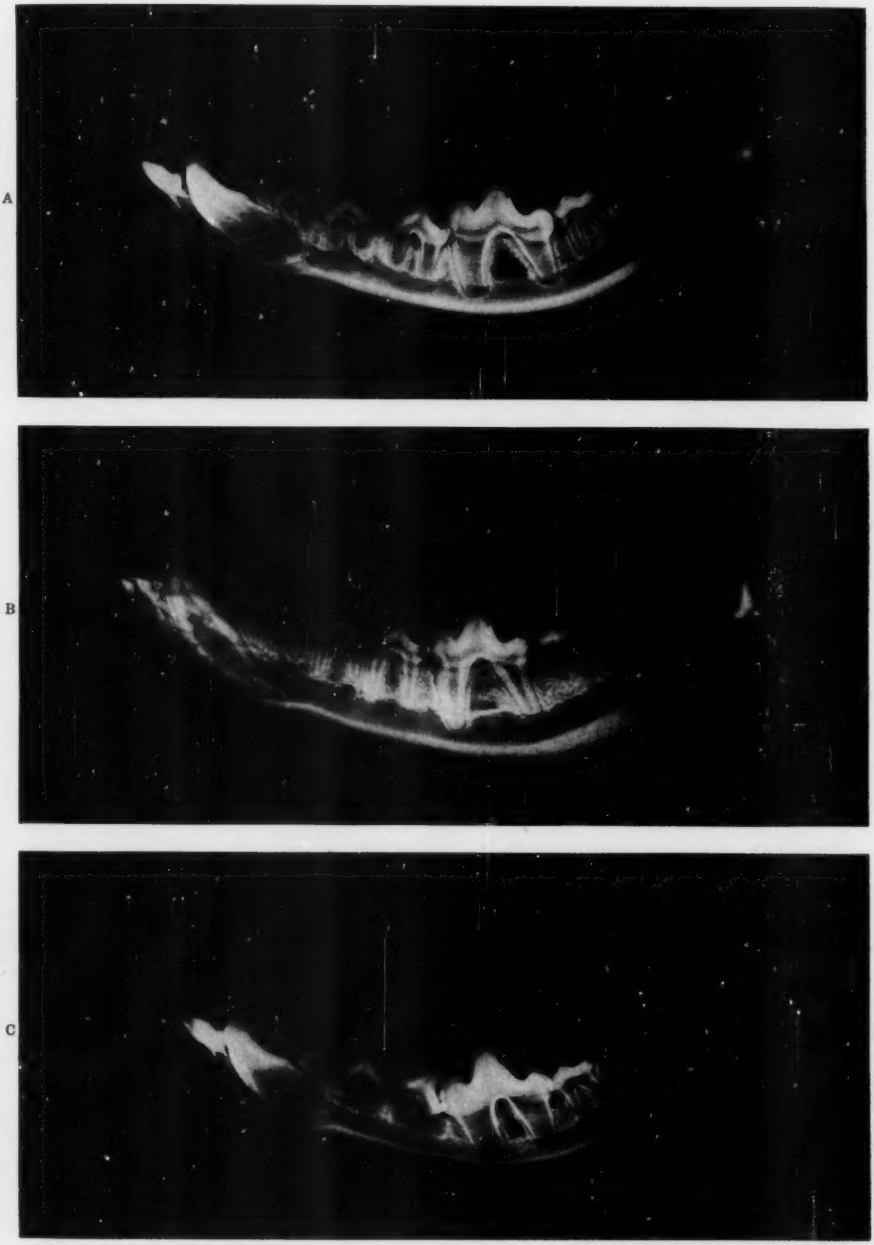


Fig. 3—The jaws of A, B and C are those of dogs 15, 14 and 13, respectively. Note radiolucency under bifurcations of molars of A and C, indicative of resorption of bone, which is characteristic for deficiencies in both filtrate fraction alone (A) and combined filtrate fraction and nicotinic acid deficiencies (C). For histologic aspect see Fig. 20.

which received no filtrate fraction, developed rampant caries, which is especially marked in dog 12. In addition, this animal developed severe periapical infections as a result of pulp involvement and shows the absence of practically all roots in upper and lower jaws. Because of this interesting phenomenon of the absence of roots, one of the upper jaws of dog 12 is reproduced in Fig. 4.

It may be noted (Fig. 2-C) that the size of the jaw of dog 11 exceeds by far those of dogs 10 and 12, which is not the result of the dietary regime, but rather of the difference in breed. The osseous structure appears hazy, or milky, with a severe marginal bone atrophy.

Fig. 4—Dog 12: Left upper jaw showing complete absence of all roots, also seen in lower jaw (Fig. 2-B).

There is no evidence of dental caries in the younger dog 15 (Fig. 3-A) which was fed the same diet deficient in filtrate fraction as dogs 10 and 12, and the paradental and gingival disturbances are far less marked in dogs 13 and 14 (Fig. 3, B and C) than in dog 11 (Fig. 2-C). It may be interesting to note that there are radiolucencies under the bifurcations of the large molars of dogs 13 and 15, and on histologic examination appear to be the result of pronounced circulatory disturbances, accompanied by a loss of bone trabeculae (*See* later).

From an experimental point of view, the material may be divided into three groups:

1. Changes accompanying filtrate fraction deficiency.
2. Changes accompanying filtrate fraction *and* nicotinic acid deficiencies.
3. Changes accompanying nicotinic acid deficiency.

And again they are subdivided into a description of changes of dental tissues, bony structure, and gingivae.

Fig. 5—Destruction of pulp tissue due to dental caries (Figs. 2-B and 6) with periapical inflammation and granulation tissue formation. Roots are absent as result of malformation (Spec. 4557, Pl. 7341).

HISTOPATHOLOGIC ASPECT

Changes Accompanying Filtrate Fraction Deficiency

From a dental point of view, the development of caries in these dogs is probably one of the most outstanding phenomena. In both animals 10 and 12, surface decay as well as deep, penetrating cavities, were found clinically and roentgenographically.

Fig. 5 shows not only complete disintegration of the pulp tissue of the molar due to caries, but also periapical inflammation and a considerable amount of destruction of osseous tissue and formation of granulation tissue.

An enlargement of the decay area in the premolar of the lower right side of dog 12 is given in Fig. 6 and the corresponding histologic slide of this severely malformed tooth is shown in Fig. 7.

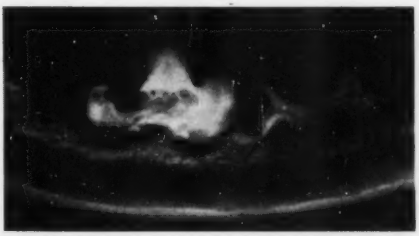


Fig. 6—Dog 12: Magnification of molar and premolars, demonstrating deep penetrating caries. For histologic aspect of premolar see Figs. 9 and 10.

X

Fig. 7—Advanced dental caries of premolar of dog 12 (Fig. 6) with osteoclastic activity deep in the dentin (Spec. 4557, Pl. 7348).

X

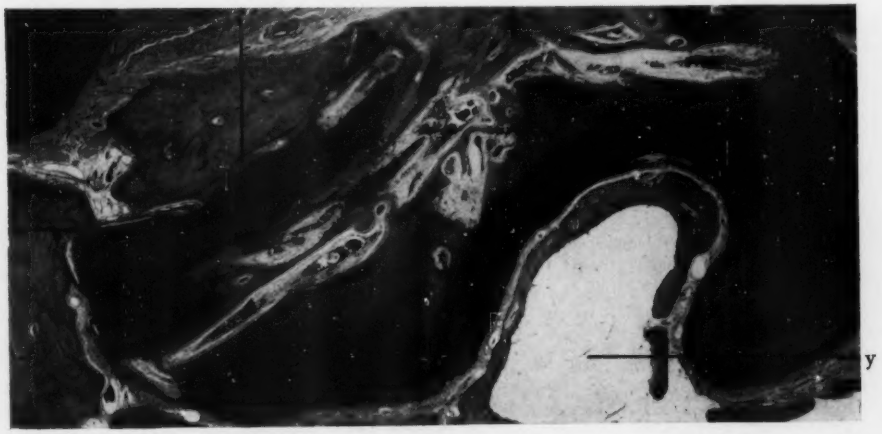


Fig. 8—Magnification of ankylosed areas (x) of premolar of dog 12. Note advanced osteoporosis (y) under bifurcation (Spec. 4556, Pl. 7345).

Severe odontoclastic activity deep into the dentine can be noted in these figures; calculus formation is very pronounced and the entire pulp tissue is changed into granulation tissue. The question as to whether the short or absent roots are the result of active resorption, or, possibly, because they were never formed under the influence of the dietary deficiency may be explained by Fig. 8.

As seen in Fig. 5, the mesial root of the first molar (a) shows no signs of earlier resorptive processes in cementum or dentine and other serial sections of anterior teeth confirm that the shortness of roots is primarily the result of malformation. On the other hand, the distal root of the premolar (Fig. 8) indicates definite resorptions, active and inactive, with ankylosis between bone and dentine structure in a large number of areas (Fig. 8). From the standpoint of osseous changes, it may be pointed out that a large number of bone trabeculae have disappeared under the shallow bifurcation of the premolar which are replaced by fat tissue (Fig. 8), showing the typical picture of osteoporosis in a comparatively young animal.

The gingival tissue of all animals on the filtrate fraction deficiency showed definite symptoms of gingivitis, as seen in Fig. 9-A.

A B

Fig. 9—A. Marked round cell infiltration of gingival margin, with partial destruction of mouth and enamel epithelium in molar area of dog 12 (filtrate fraction deficiency). (Spec. 4550, Pl. 7334).
B. Inflammatory reaction is missing in control dog 4 (Spec. 4522, Pl. 7351).

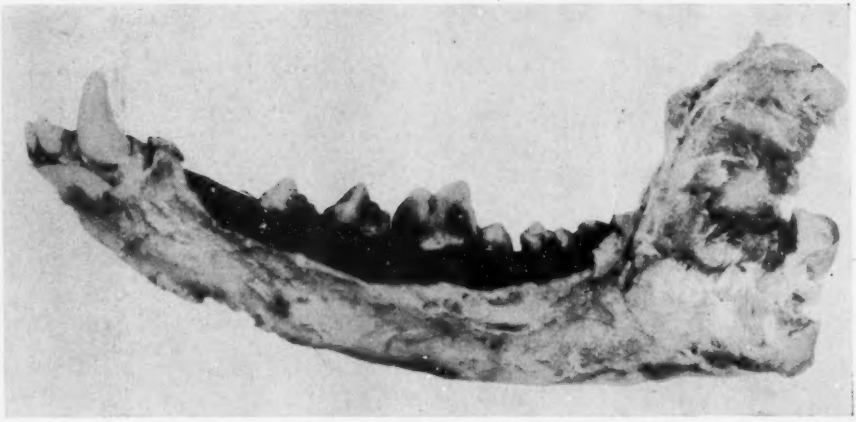


Fig. 10—Left lower jaw of dog 11 after a period of 343 days on a diet deficient in filtrate fraction and nicotinic acid. Severe gingival and paradental infections are accompanied by marked marginal atrophy. The bifurcations of most molars and premolars are involved.

Round cell infiltrations and destruction of the enamel epithelium are very marked in the experimental animal (Fig. 9-A), while they are completely missing in the controls (Fig. 9-B). The severe hypercementosis at the cementoenamel junction in Fig. 9-A is an observation made frequently in hypervitaminosis D (1) and must be considered as being unrelated to the filtrate fraction deficiency. Since this animal was not weaned in the laboratory, the possibility exists that a large dosage of vitamin D before the beginning of the experiment is responsible for it.

Changes Accompanying Deficiencies in Filtrate Fraction and Nicotinic Acid:

Two dogs were fed these deficient diets for 150 and 343 days, respectively. As stated previously, the younger dog, 13, did not develop any outstanding pathologic bone changes because the experimental period apparently was too short. However, clinically, all characteristic symptoms of a "black tongue" were noticed at the end of the experiment. On the other hand, the changes produced after a longer experimental period in dog 11 were most remarkable.

Fig. 11—Roentgenogram of last premolar and first molar of dog 11 (Fig. 15). Marginal atrophy throughout lower jaw is most marked. Osseous structure appears irregular; trabeculae have lost their sharp and clearly-defined character. This picture shows all symptoms of a general mandibular osteitis.

Fig. 10 shows the morphologic aspect of the left lower jaw of dog 11, with severe marginal alveolar resorption and calculus formation, while Fig. 11 gives the roentgenographic aspect of the molar and premolar region. The osseous structure does not appear clearly defined, but rather hazy, or "milky." The marginal bone atrophy has passed the bifurcations and clinically these teeth were severely loosened. It may be mentioned here that Fig. 11 emphasizes the importance of the highest standard of roentgenography in order to be able to see early and detailed structural changes. While the dental structure apparently did not suffer any effects through this deficient diet, the gingival tissue and the bony structure show clinically and histologically severe inflammatory reactions.

All free margins of the gum, without exception, show most extreme infectious processes, with proliferation of the entire epithelium, round cell infiltration and granulation tissue formation in the presence of deep pockets (Fig. 12). Clinically, the gum tis-

sue looked dark, red and puffy, with large amounts of soft and hard deposits on the surfaces of the teeth. These inflammatory reactions are not only visible around the teeth, but the entire mucous membrane of the oral cavity had the same appearance.

Fig. 12—Gingival tissue of dog 11 is severely inflamed, with marked proliferation of mouth and enamel epithelium. The inflammation reaches deep into the paradental structures (Spec. 4554, Pl. 7335).

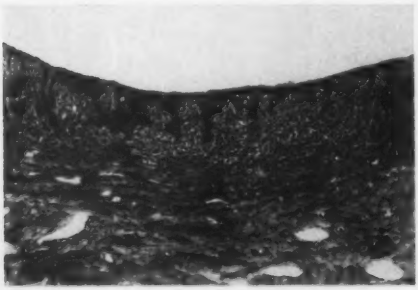


Fig. 13—Same infiltration of subepithelial connective tissue as seen near the teeth (Fig. 12) can be found anywhere in the oral cavity. This section is taken from the mucous membrane of the cheek (Spec. 4554, Pl. 7336).

Fig. 14—Dog 11: The inflammatory process of the gingival tissue has reached deep into the paradental structure of lower premolar, accompanied by marked bone resorption. The atrophy has passed the bifurcation and extensive granulation tissue (y) has formed. The marginal peridental membrane space is widened through progressive resorptive processes (Spec. 4554, Pl. 7339).

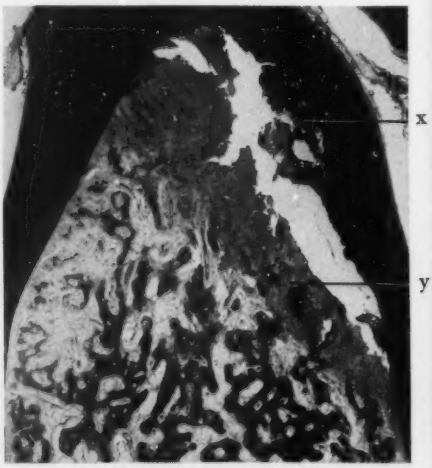


Fig. 15—Dog 11: Formation of granulation tissue (y) and heavy calculus (x) under bifurcation of lower molar, accompanied by a typical osteitis (Spec. 4554, Pl. 7340).

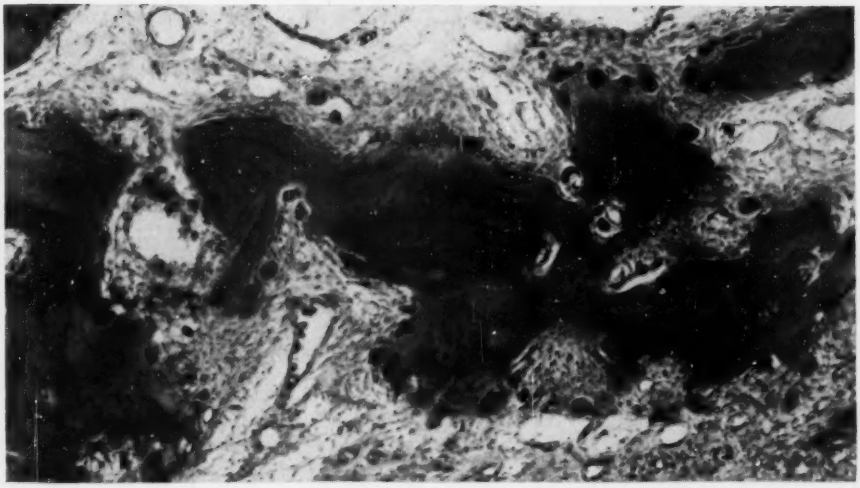
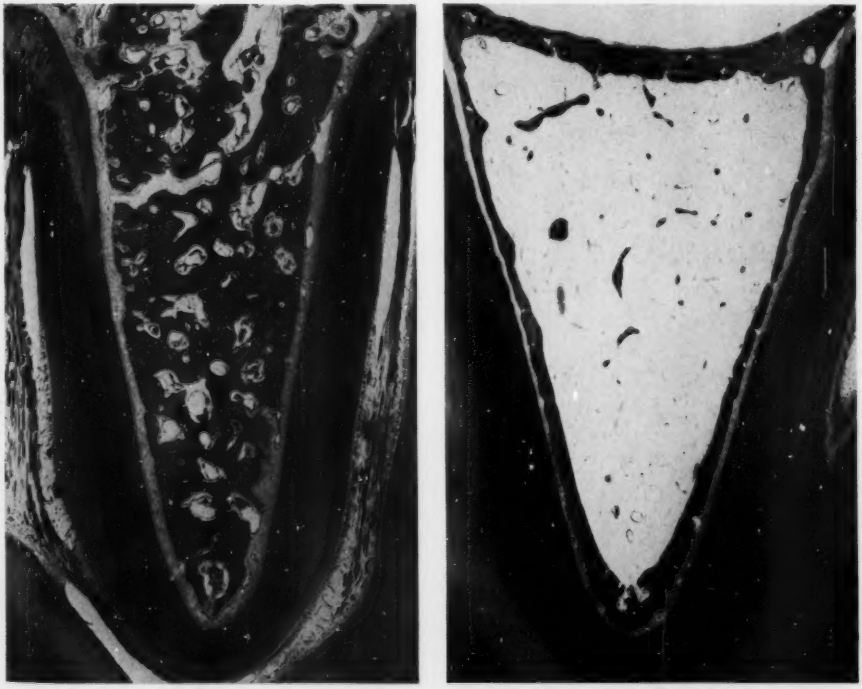


Fig. 16—Magnification of an area of Fig. 20, demonstrating marked bone resorption with a large number of osteoclasts which is characteristic for the osteitis produced by this vitamin deficiency (Spec. 4554, Pl. 7362).



A B

Fig. 17—A, Normal osseous structure under bifurcation of an upper premolar of control dog 4 (Spec. 4522, Pl. 7346).
B. Marked osteoporosis with almost complete loss of all bone trabeculae in upper jaw of dog 11. Fat tissue has replaced the osseous structure (Spec. 4554, Pl. 7353).

This may be demonstrated in Fig. 13, which is a specimen taken from the cheek tissue.

Central histologic sections taken through the bifurcation of a premolar, as seen in Figs. 14 and 15, show that the infection reached deep into the jaw bone structure, presenting the typical picture of an osteitis.

Here we find (Figs. 14 and 15) recession of bony tissue, with heavy calculus (x) under the bifurcation and granulation tissue formation (y). Replacement of the newly-formed osseous structure is apparent in certain areas, while, in general, osteoclastic activity dominates the picture, as seen in Fig. 16.

Fig. 18—Dog 11: Advanced osteoporosis of interproximal bone septum, with complete loss of "supporting bone," which is replaced by fat tissue 4554, Pl. 7343).

Changes in the upper jaws of dog 11 appeared to be of a different type. Even though the gingival tissues showed the same inflammatory reactions, perhaps to a lesser extent, the osseous changes are more of a porotic nature, which may be seen in Fig. 17.

Here we find in Fig. 17-A the normal distribution of bone trabeculae under the bifurcation of the premolar of a control dog, while in the nicotinic acid-deficient dog in Fig. 17-B, the bony support (in fact, we call it "supporting bone") has been taken out completely by the deficient diet. Only a few fragments of bone trabeculae are left and communications between the marrow cavity and the peridental membrane space are evident. The osseous structure is replaced by fat cells and the osteoclasts are continuing their work on the rather thin alveolar bone.

The capillaries are tremendously enlarged throughout the entire osseous structure, which is in contrast to the normal bone picture. The same picture of osteoporosis is found throughout the entire upper jaw under bifurcations as well as in the interproximal bone septa, as demonstrated in Fig. 18. The round cell infiltration of the subepithelial connective tissue is less marked.

In view of the fact that the younger dogs did not show the marked clinical and histologic changes observed in the older group, an attempt was made to find in these dogs which early manifestations might be responsible for the resorption of osseous structure, leading to osteoporosis and a possible inflammatory predisposition. Roentgenographically, the areas under the bifurcation of premolars of dogs 13 and 15 showed pronounced radiolucencies (Fig. 3).

In Figs. 19 and 20, which were taken of the premolar areas of dog 15, tremendously increased circulation throughout the entire osseous structure and also pulp tissue can be seen. There are innumerable hemorrhages in the odontoblastic layer and pronounced osteoclastic activity near the enlarged blood vessel. The central portion of the bone structure under the bifurcation (Fig. 20) has been resorbed and replaced by fat tissue. The enlargement of all capillaries in this very young osseous structure and also in the pulp is most pronounced.

It may be stated here that dog 11 was on a diet deficient in filtrate fraction *and* nicotinic acid and that the rather marked inflammatory tendency of the gum tissue with a subsequent involvement of the osseous structure (not noted in the filtrate fraction deficient animals) must be explained as being primarily the result of the missing nicotinic acid. The marked osteoporosis in these comparatively young animals, however, may be seen separately in both groups. This confirms previous observations that the rather unstable osseous structure may undergo marked resorptive changes by a large number of systemic factors. In other words, the osteoporosis is by no means a nosologic entity.

Changes Accompanying Nicotinic Acid Deficiency:

There was only one young dog on this deficiency for approximately eight months (dog 14). The gingival tissue shows severe inflammatory reaction similar to the filtrate fraction and nicotinic acid-deficient dog 13. The experimental period apparently was not sufficient to produce more specific changes which may point out that the period of deficiency plays an important role in bringing about changes as they were observed in the older dog 11. No bone or dental changes were seen in contrast with those observed in dogs 10, 11 and 12. These gingival changes are sharply reminiscent of those observed in canine black tongue which is ascribed to nicotinic acid deficiency.

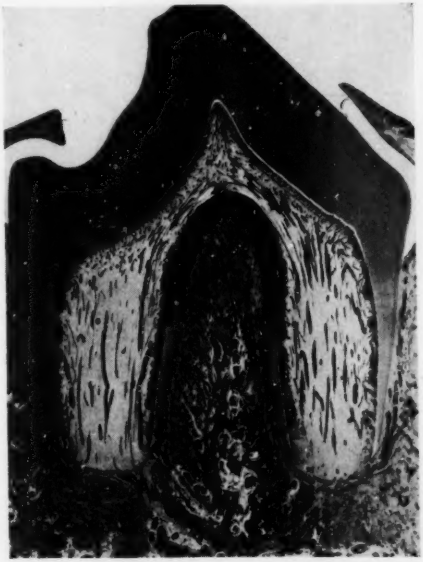


Fig. 19—Dog 15: Markedly increased circulation with perivascular hemorrhages in marrow cavities and pulp tissue (Spec. 4565, Pl. 7342).

Fig. 20—Dog 15: Magnification of area under bifurcation of molar, showing tremendous enlargement of all capillaries and resorption of bone (Spec. 4554, Pl. 7343).

SUMMARY AND CONCLUSIONS

Clinical observations of beneficial effects of vitamin B preparations on inflammatory conditions of the oral cavity and in particular paradental tissues have pointed out that vitamin B deficiencies are important etiological factors in the production of the inflammatory type of paradentosis. From these investigations in dogs, the following conclusions can be drawn:

1. The filtrate fraction of the vitamin B complex and nicotinic acid must be recognized as essential food accessories for the health of dental, gingival and paradental structures because their absence leads to severe gingival inflammation and infection of the underlying osseous structure. The clinical as well as the histologic aspects resemble those of the "inflammatory type" of para-

dentosis observed in human beings with marked calculus formation.

2. In areas where the inflammation has not reached the paradental bone structure, a typical osteoporosis results, with severe enlargement of all of the smallest capillaries, increased osteoclastic activity and replacement of bone by fat tissue.
3. Filtrate fraction deficiencies alone lead to severe malformation and resorption of roots, as well as resorption of paradental bone structure, resulting in varying degrees of osteoporosis.
4. Filtrate fraction deficiencies also lead to rampant decay in young dogs, which are otherwise rarely observed.
5. Nicotinic acid deficiency alone appears to lead to gingival disturbances, but not to dental or osseous changes such as were observed in those animals which suffered both filtrate fraction and nicotinic acid deficiencies simultaneously, or filtrate fraction deficiency alone. The black tongue of nicotinic acid deficiency may represent an exaggerated form of the changes here seen.

In general, it may be stated that this first experimental evidence of a relationship between vitamin B fractions and dental and paradental disorders herein presented offers new hope for a successful treatment of these conditions in human beings by the addition of systemic therapy.

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Diffuse Atrophy of the Alveolar Bone (Periodontosis) *

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INTRODUCTION AND REVIEW OF LITERATURE

In 1920, Gottlieb[1] first described the clinical and histopathological picture of a non-inflammatory disease of the supporting tissues of the tooth. He termed it, "diffuse atrophy of the alveolar bone." In 1923,[2] he added essential findings to his initial report. He claimed that the atrophy of the alveolar bone, and the widening of the periodontal membrane, are the chief characteristics of this disease. At the same time a resorption of the cementum can take place. The development of a deep gingival crevice, with an inflammatory reaction, is a secondary factor. Clinically, the first symptom is migration of the affected tooth. Whereas, in the anterior teeth migration is, usually, the earliest symptom, in the posterior region loosening of the bicuspid and molar teeth,—where the interlocked cusps prevent free movement,—is noticed first. Gottlieb claimed that it is not the external damaging factors that determine the future of the dentition, but rather the potentialities of cemental repair. He described a diffuse atrophy in a case of epidemic influenza. The gingival tissues showed no marked pathological changes, and were average for the age of the patient. However, there were extensive resorptions on the apices of all teeth. The periodontal space was widened at the apices, due to resorption of the cementum and alveolar bone. The periodontal membrane had lost its functional structure and had changed into loose connective tissue. At the same time Gottlieb demonstrated cementum repair following root resorptions, and interpreted these as spontaneous healing of cemental damage.

H. K. Box[3] differentiated between "simplex" and "complex periodontitis." The "complex" form, clinically described, closely resembles Gottlieb's "diffuse atrophy." Box described the histopathological changes, which take place in the periodontal structures, as "rarefying pericementitis fibrosa," and classified them as inflammatory lesions. As a cause of this process, Box merely referred to overstress in the following statement: "Overstress, in the pericementum, is the physical agent; overstrain, the state produced by it, and 'rarefying pericementitis fibrosa,' a resultant lesion."

In the literature of the past twenty years, only a few cases have been described, giving additional information. One of these cases was investigated and the report published by E. Mueller, H. Rony, and R. Kronfeld.[4, 5, 6] It was that of a woman, aged 36 years, who had developed several rapid resorptions on her teeth and, ultimately, lost some of them. The patient's condition was thoroughly investigated roentgenographically, medically, and histopathologically. The case was unusual and, at the time of investigation, it was recognized as an exceptional form of "diffuse atrophy." The medical examination revealed general neurasthenia, vasomotor instability, and functional disturbance of the liver, the cause of which was unknown. Medical treatment was successful, and tooth resorptions not only ceased, but repair processes set in, as roentgenographic and histopathological investigations have shown.

Another specimen was described by Gottlieb and Orban, in their publication on gingival inflammation and loosening of teeth.[7] This case will be discussed, together with some recent findings.

Kurt H. Thoma and Henry M. Goldman[8] corroborated fundamentally Gottlieb's conception of periodontal diseases and, especially, of diffuse atrophy. They described a case, in advanced condition, in which the bone resorption was followed by epithelial proliferation and deep pocket formation. Thoma and Goldman considered the primary pathological changes in diffuse atrophy as non-inflammatory.

From the review of dental literature it is evident that more cases of this type of periodontal disease will have to be investigated to reach a satisfactory conclusion. Where the condition had advanced extensively, no information seems to be available as to the course of the disease, secondary processes having obscured the primary picture. The study of cases, in their earliest

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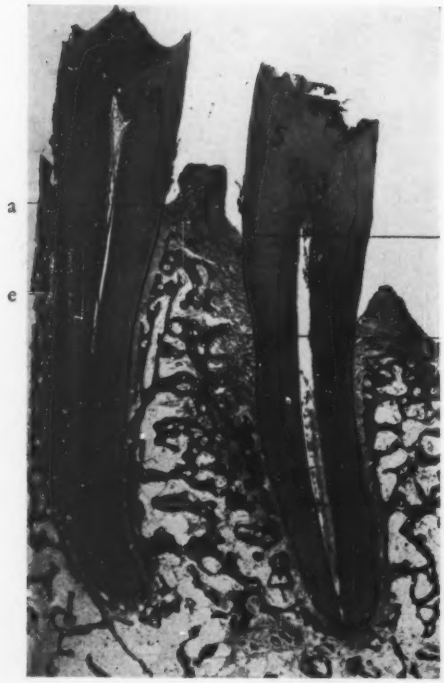

Fig. 1—Lower bicuspids of a case of unknown age. The gingival crevices in the interdental space (a-b) are on the cementum. The periodontal space, in the gingival portion (c), is extended on the mesial side of the second bicuspid. The rest of the periodontal membrane is of normal width. The bone marrow on the mesial side of the second bicuspid is fibrous and edematous (d). (e)—mesial side of first bicuspid.

stages, promises useful information as to the development of this disease.

RECENT OBSERVATIONS

Some instructive observations were made in the investigation of the microscopic specimens of a human jaw (the age of the case being unknown) where diffuse atrophy was diagnosed before histological preparation. Fig. 1 shows the interdental space between the left lower bicuspids. The lower molars and upper second bicuspid and first molar were missing on this side. The lower second bicuspid had drifted slightly to the distal. The occlusion is shown in Fig. 2, which is a diagrammatic drawing from photographs. The photomicrograph (Fig. 1) shows a considerable widening of the gingival part of the periodontal space, on the mesial side of the second bicuspid. At the apical part of the root the periodontal membrane is of normal width; so is the entire periodontal membrane of the first bicuspid. The interdental papilla shows slight chronic inflammation, with acute symptoms close to the bottom of the gingival sulci. On both teeth (Fig. 3) the epithelium is attached to the cementum. Part of the root surface is exposed, and

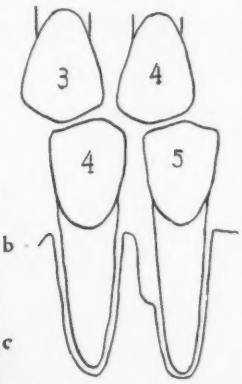

Fig. 2—Diagram of the occlusal relationship of the cuspid and bicuspids of the case in Fig. 1.

Fig. 3—Interdental space of the two lower bicuspids of Fig. 1: (a)—interdental papilla. (b)—periodontal membrane of normal width on the distal side of the first bicuspid. (c)—widened periodontal space on the mesial side of the second bicuspid. (d)—normal width of the periodontal membrane in the apical portion of the mesial side of the second bicuspid. (e)—epithelial attachment. (f)—epithelial island attached to the surface of cementum.

Fig. 4—Higher magnification of the region (c) in Fig. 3. Degeneration of the connective tissue (a) of the suspensory apparatus; (b)—cementum; (c)—bone.

there is a slight amount of calculus in the sulci. The marginal inflammation does not penetrate into the periodontal membrane on the first, or second bicuspid. The principal fibers of the periodontal membrane, in the widened region, appear to be disarranged. In some places, the fiber bundles are attached only to the cementum (Fig. 4). They do not extend from the cementum to the bone but end, intermingled, in loose connective tissues. Other fiber bundles are attached only to the bone trabeculae, but they end loosely—brushlike, in the loose connective tissue, between the deranged remnants of dense principal fiber bundles which seem to have dissolved. In this fluid-like tissue, single, scattered, migratory cellular elements are found without forming any infiltrations. The widening of the periodontal space must have taken place some time previous to death, as only very few areas of active bone resorption can be observed in this region. The bone trabeculae, generally, show signs of inactivity or only slight new formation.

In the more apical regions of the periodontal membrane similar tissue degeneration can be observed. Fig. 5 shows one of these areas. The periodontal membrane is extended over a circumscribed area, whereas, in the neighboring regions, it is of normal

Fig. 5—Higher magnification of the area (d) of Fig. 1; (another section of the same series) (a)—edematous loose fibrous tissue, with numerous capillaries in the widened periodontal membrane, extending into the bone marrow. The rest of bone marrow is fatty (b); c—normal periodontal membrane.

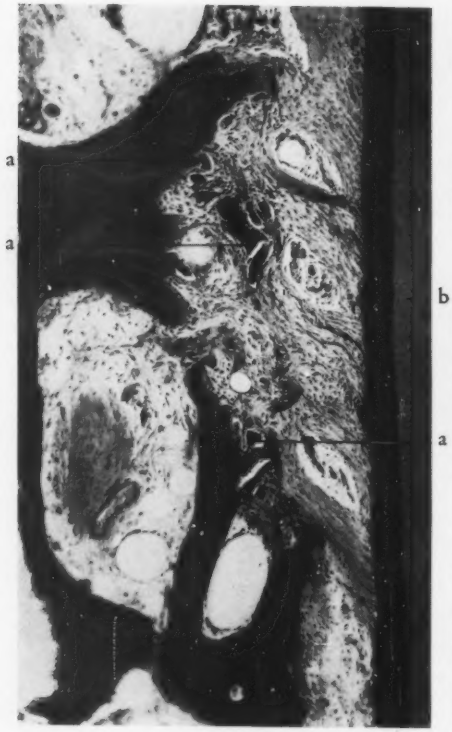

Fig. 6—Mesial alveolar margin—(e) of Fig. 1—first bicuspid. Resorption of the alveolar bone (a) due to traumatic pressure; (b)—mesial surface of the tooth.

width and structure. In the widened region, the principal fibers of the periodontal membrane are mostly missing and are replaced by a loose network of fibrous, seemingly edematous tissue, without inflammatory reaction. Numerous branching thin capillaries are found in the tissue and, in some sections, they resemble a brushlike arrangement. The bone marrow, adjacent to the first bicuspid, is formed by fatty marrow (Fig. 3), whereas, the bone marrow which is close to the second bicuspid, is filled with a loose fibrous tissue, similar to that found in the widened parts of the periodontal membrane.

Following up the changes which took place in the supporting structures of these two bicuspids, conditions were found which indicate traumatic influence on both teeth. The first bicuspid was pushed in a mesial direction by its antagonist, tipping the tooth slightly toward the mesial side. Due to this

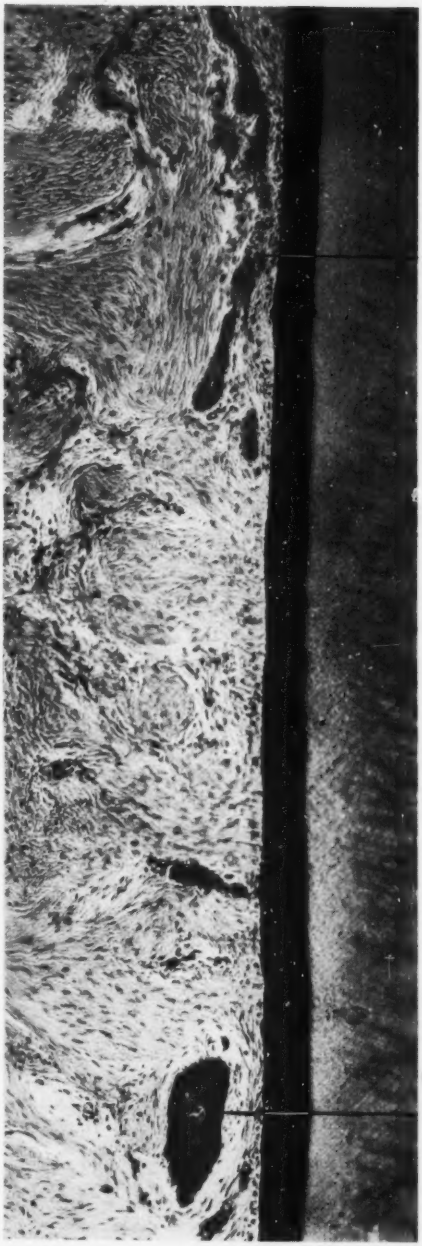

Fig. 7—Proliferated epithelial rests in the periodontal membrane of the second bicuspid. (a)—epithelial rests grouped in cords and buds; (b)—large island with hornification in the center.

pressure, resorption was found, as shown in Fig. 6. The line of the alveolar bone is broken by resorption lacuni, filled with osteoclastic cells. The adjacent bone marrow was somewhat edematous and fibrous, but no signs of cellular infiltration were noticeable. Fig. 6 shows typical pathological changes, due to traumatic injury in the periodontal membrane. These changes are identical with those found in experimental traumatic occlusion.[9] On the distal side of this first bicuspid (Fig. 1), the principal fibers in the periodontal membrane are taut and close to the margin of the alveolar bone and, in several places, bone formation is in progress. Traumatic changes could be found also at the apex and distal side of the second bicuspid, indicating axial and distal pressure.

Not only the connective tissue elements showed striking changes, but also the epithelial structures of the periodontal membrane. The epithelial rests were enlarged and seem to have proliferated. In Fig. 7, two islands of epithelial remnants can be observed. In one, the epithelium is grouped in cords and buds and, in the other, a large epithelial island has formed. Whereas the epithelium in the cords is composed of only two or three layers of cells, many layers are present in the island. The cells of the outermost layer of this island are cuboid in shape and represent the germinative layer. Toward the center there are prickle cells and, in the central area, the cells seem to be hornified. Such proliferated epithelial islands could be found scattered in all specimens of the periodontal membrane in which they lie, separated from the cementum by fibroblasts, fibers, and cementoblasts.

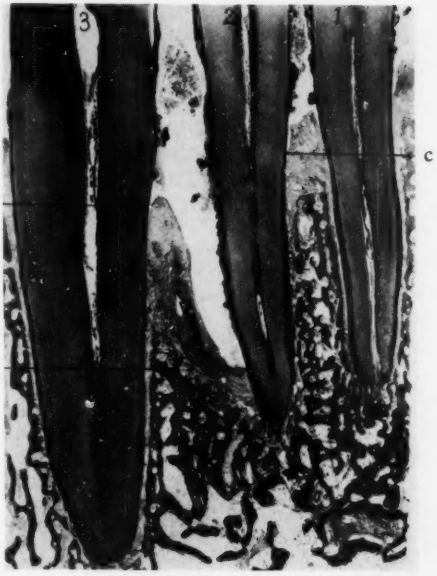

Fig. 9—General view of the lower left central (1) and lateral (2) incisors and cuspid (3) of the same case as shown in Figs. 1-8; on the distal side of the lateral incisor is a deep gingival crevice (a); on the adjacent surface of the cuspid the epithelial attachment. (b) is on the gingival third of the root. On the mesial side of the lateral and central incisor the epithelial attachment is in the gingival third (c).

Fig. 8—Higher magnification of area (f) in Fig. 3. Isolated epithelial attachment (a) on the surface of the cementum.

Another finding, concerning epithelium in the periodontal membrane, could be observed in this specimen: On the surface of the cementum, in the enlarged part of the periodontal membrane, was an epithelial island, attached to the cementum. This island (Fig. 8) corresponds to f in Fig. 3. The epithelium is separated from the marginal epithelial attachment by connective tissue; connection could not be traced in any of the available specimens. The island consists of from three to five layers of epithelial cells which are connected by intercellular bridges

and lined with a germinative layer. The possible significance of this finding will be discussed in connection with other observations concerning the epithelial attachment.

Other pathological conditions were found which are significant in this disease. Fig. 9, shows the lower left central and lateral incisors and the cuspid. There is a deep gingival crevice on the distal surface of the lateral incisor. There is no evidence of deep pocket formation on the mesial side of this tooth nor around any of the other teeth. Whereas, the epithelial attachment is, in all areas, comparatively close to the cementoenamel junction (leaving some of the cementum exposed), the epithelial attachment, on the distal side of the lateral incisor, is almost at the apical foramen. The alveolar bone follows the outline of the gingiva. On the mesial side of the lateral incisor, the alveolus covers about two-thirds of the root surface; at the distal side, the entire alveolar bone is resorbed. On the mesial side of the cuspid, the alveolar bone covers about half of the root, but at the distal side of the same tooth, at least five-sixths of the root surface is attached to the alveolar bone.

Calculus is deposited in the gingival crevices and food has lodged in the interdental spaces. Generally, the gingivae show only slight inflammatory reaction, whereas, between the lateral incisor and cuspid, the gingiva is considerably inflamed. The epithelial covering is missing in several places, thus exposing connective tissue. Where epithelium is present, it is very thin and infiltrated with leukocytes.

The periodontal membrane of all teeth, including the lateral incisor, is free from any inflammatory infiltrate. The bone marrow, bordering the deep pocket, is edematous and fibrous in character. It shows inflammatory cells in the surface areas, but, otherwise, the marrow is of a fatty, normal nature. The arrangement of the bone trabeculae, around the affected tooth, as well as around the other teeth, shows no deviation from the normal.

In the jaw of another case, aged 55 years, a diagnosis of diffuse atrophy (periodontosis) was made previous to its preparation for microscopic study. The two right upper bicuspids of this case are shown in Fig. 10; both teeth are out of occlusion; the first bicuspid, apparently, for a shorter period than the second bicuspid. On the distal side of the second bicuspid is a periodontal abscess (lateral or parietal abscess) which has been described in a publication by Gottlieb and Orban.[7] The specimen was also used by J. P. Weinmann[10] in his study on the progress of inflammation into the supporting structures. In the section, shown here, no connection between the pocket and abscess can be observed. However, by following up the serial sections, the connection could be clearly detected. It was a pocket which did not follow a straight, but rather a spiral course.

Between the two bicuspids the interdental papilla is considerably inflamed. It shows a deep pocket on the distal side of the first bicuspid, and a comparatively shallow pocket on the mesial side of the second bicuspid. The outline of the tartar, above the papilla, and in the gingival crevice, indicated the extent of shrinkage, due to histological preparation. Both pockets show calcific deposits at the bottom. The epithelial wall of the deep pocket is broken in several areas, permitting an easy flow of leukocytes. A higher magnification of the interdental papilla, Fig. 11, shows the accumulation of inflammatory cells, along the wall of the pocket and, especially, close to its bottom. The inflammation is severe on the side of the first bicuspid pocket, but only slight on the mesial side of the second bicuspid. Transseptal fibers limit the field of inflammation. Along the blood vessels, some inflammatory reaction can be followed into the bone marrow spaces. The mesial side of the first bicuspid (Fig. 10) shows a very deep gingival crevice, considerably deeper than on any of the other surfaces of the teeth.

A finding, previously not discussed, was observed on the mesial side of the second bicuspid in the interdental space. Close to the opening of the periodontal membrane, a slight thickening of the cementum has occurred. Above the point, where the cementum hyperplasia sets in, an epithelial cord, attached to the cementum, can be seen (Fig. 12). In the sections, available for study, no connection between the marginal epithelial attachment and the epithelial cord could be found. It can be assumed that the epithelial cord is the end of an epithelial attachment which proliferates around the tooth from the distal or buccolingual sides. Between the end of the marginal epithelial attachment and epithelial cord, connective tissue fibers are attached to

the cementum, and only slight inflammatory infiltration is present between the fiber bundles. At about the level of this epithelial attachment, the principal fibers of the periodontal membrane are partly broken down and separated by edema. In some of these areas very few migratory cells can be seen but, in other areas, they have gathered in large numbers.

DISCUSSION

According to *clinical* observations, the earliest symptom of diffuse atrophy is migration of teeth. This occurs without inflammation in the periodontal tissues. Subsequently, the teeth loosen; pocket formation and inflammation are secondary occurrences.

The *histo-pathological* findings, grouped according to the sequence of their occurrence, correspond to the clinical sequence. Three different stages can be recognized:

In the *first stage* degeneration of the principal fibers of the periodontal membrane occurs (Fig. 4) with a localized widening of the periodontal membrane (Fig. 3) due to resorption of the alveolar bone. During this process a proliferation of the capillaries can be observed (Fig. 5) with the development of a loose connective tissue. There is no inflammation in this stage and no proliferation of the epithelial attachment.

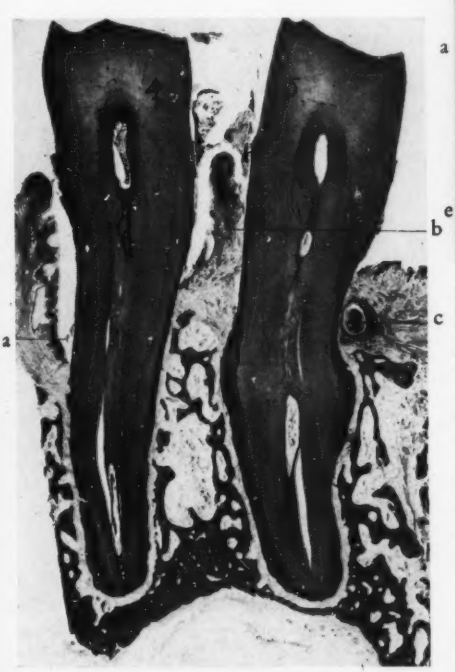

Fig. 10—General view of upper bicuspids of a case, 55 years of age. Deep pocket (a) on the mesial side of the first bicuspid; (b)—interdental papilla between the two bicuspids: (c)—periodontal abscess on the distal side of the second bicuspid.

Fig. 11—Higher magnification of the interdental papilla (b) of Fig. 10. Deep crevice (a) on the distal side of the first bicuspid. Shallow crevice (b) on the mesial side of the second bicuspid; end of the epithelial attachment (c); isolated epithelial attachment (d); alveolar crest (e).

The *second stage* is characterized by the proliferation of the epithelial attachment along the root surface (Fig. 11). During this period there is a slight cellular infiltration in the connective tissue. These scattered cellular elements are of the plasma cell, and polyblast type.

Fig. 12—Higher magnification of the isolated epithelial attachment (d) in Fig. 11; (a)—end of the epithelial attachment; (b)—chronic inflammatory reaction between fiber bundles.

In the *third stage* the epithelium of the proliferated attachment separates from the surface of the root, and deep gingival crevices develop (Fig. 9). The inflammation increases due to irritation and infection from these deep crevices.

First Stage

According to clinical experience, the first stage in the development of this disease is short. Secondary changes, especially inflammation, set in and obscure the primary conditions. Therefore, the first stage is seldom clinically observed or recognized. It is even more difficult to obtain an autopsy or biopsy specimen of this stage. It was fortunate that an example was found of this condition (Fig. 1-5), not as yet obscured by secondary changes.

The most characteristic feature of the pathological changes, in the first stage, is the disorganization of the principal fiber apparatus of the periodontal membrane. The fiber bundles which, under normal conditions, are stretched between the cementum and bone in an oblique direction, appear to be unattached at one end or the other. Between the remnants of the fiber bundles a hydropic, loose structure can be observed. Some of the fibers, within a fiber bundle, show a type of granular destruction. This gives the impression that destruction of the connective tissue fibers leads, gradually, to a hydropic degeneration.

These changes resemble pathological conditions in other parts of the body where connective tissue has a similar function to that of the periodontal membrane. Such connective tissue is found in tendons, ligaments, and elsewhere where collagenous fibers are the predominant tissue element. Several reports have been made on the degenerative processes of such tissues.[11] All authors agree that the primary changes in the connective tissue fibers are of a degenerative character and that there is no accompanying inflammatory reaction. These degenerative changes are: loosening or swelling of the interfibrillar substances and granular disintegration of the fibers. In more advanced cases the tissue may undergo vacuolization and hydropic degeneration. This process is confined to circumscribed areas. In general pathology this entire process is termed "desmolysis," i. e.: dissolution of connective tissue. As to the causes of this degenerative process, there is no agreement among the authors. Some are inclined to blame traumatic influences; others claim a toxic origin, or both.

Since the tissues of the periodontal membrane belong, structurally as well as functionally, to the same tissue classification as tendons and ligaments, the supposition is justified that pathological conditions in the entire tissue group are comparable. Differences are to be expected, of course, because of the special nature and function of the dental organ. The absence of inflammatory reaction, in the first stage of diffuse atrophy, as shown in Figs. 3, 4, and 5, demonstrates a further similarity in the pathogenesis of the degenerative processes of these tissues.

It is interesting that one author[12] made the observation that a degeneration of tendons and ligaments occurs in 71% of females, between the ages of ten and twenty-five, whereas males are, generally, not affected until after the age of thirty. The age distribution of this disease, in females and

males, seems to be another parallel to periodontosis.

The causes of the degeneration of the fibers of the periodontal membrane are as nebulous as those of other tendons and ligaments. It has been claimed that toxic and traumatic influences play a part in both instances. In the case of periodontosis, inflammation was considered an additional primary cause. This conclusion was reached because the earliest stage is very seldom clinically observed and has not as yet been seen at the gingival margin in a microscopic specimen. In Gottlieb's case,[2] the disturbance set in at the apex of the teeth, whereas the marginal tissues were normal. The case, presented here, proves indubitably that inflammation is not a primary factor in the development of the disease.

In considering other possible causes, trauma must be eliminated as a causative factor for several reasons. Pathological changes, produced by trauma, are well-known and well-defined as a result of experimental autopsies. Gottlieb and Orban[9] have shown, in their systematic experiments on dogs, that trauma produces compression of the connective tissue, bleeding, necrosis, and bone resorption on the side of pressure, with new formation of bone along the taut fibers on the side of tension. If the trauma ceases the traumatized tissues become resorbed and are replaced by normal tissues. These findings have been confirmed by different authors who used human autopsy material. In addition to these findings, cases of cusp interference, or so-called "jiggling," have been observed, showing the typical pathological changes produced by pressure on both sides of the tooth. This results in a widening of the periodontal membrane.[18] All these findings have not the least similarity to the conditions which are characteristic of periodontosis. From the point of view of pathogenesis both diseases are well defined. Traumatic changes can be observed in the case described in Fig. 6, on the distal side of the second bicuspid, and at the apex. Similar findings can be observed on the mesial side of the first bicuspid.

The clinical course in periodontosis is quite different from that in trauma. Periodontosis often occurs in complete dentition, with perfect occlusion, and no signs of caries. It might affect teeth without occlusion. On the other hand, traumatic changes are regular findings in cases where teeth are missing, migrated, or irregular in their position.

Thus, trauma and inflammation can be eliminated as causative agents.

Toxic and other systemic factors remain for consideration: There are many indications, from previous investigations, showing that systemic factors and toxic influences play an important part in the development of this disease. Gottlieb,[2] describing his first case of diffuse atrophy, considered influenza to be the probable toxic causative agent. Weinmann,[14] while investigating the specific-dynamic effect of protein in cases of diffuse atrophy, advanced the opinion that a hypofunction of the pituitary gland, or a disturbance of the autonomous nervous system, may accompany this disease entity. Mueller and Rony[4] described a case where the dysfunction of the liver was accompanied by progressive root resorptions. Boenheim[15] considered multi-glandular endocrine dysfunction as an accompanying factor. Becks[16] has shown that, in approximately 60% of his cases of this disease, a hypofunction of the thyroid gland was found and, in the remaining percentage of cases, other endocrine dysfunctions were noted. Rowe Smith[17] observed secondary anemia and endocrine disturbances in many of his cases of periodontosis. Toxic influences, produced by faulty digestive function, has been thought to be one of the important responsible agents, by M. Karolyi.[18] Unbalanced and deficient diets were responsible in one case of Gottlieb and Weinmann[19] and, in other cases, of Boyle,[20] Thoma and Goldman.[8]

This review of observations indicates that systemic influences on the periodontal structures may be manifold. It is impossible to say that any one factor is responsible for all cases because each may have a different systemic toxic background.

In the attempt to understand what part these systemic toxic factors play in the development of degenerative processes in the periodontal membrane, ultimately leading to periodontosis, it has to be concluded that a normal metabolism is necessary to ensure a continuous physiological replacement of tissue elements. It is known that all cellular elements have a certain life span and that they must be continuously replaced, especially, if they are under heavy functional influences. This, particularly, applies to the

periodontal membrane connective tissue which is continuously exposed to wear and tear. The newly formed fibers have to be attached to the cementum on one side, and to the alveolar bone on the other side, in order to fulfill their purpose. Fibers can only become attached to the cementum and bone by being embedded into the matrix of these tissues, when they are newly formed. This physiological process of newly embedded fibers, by newly formed cementum, was considered by Gottlieb[2] to be the most important factor to ensure the function of a tooth. It should be understood that the potentiality of cemental repair depends on the condition of the periodontal connective tissue. If this physiological process of regeneration is impaired by systemic-toxic influences, the degenerated fibers are not replaced and, gradually, the first stage of periodontosis develops. If the principal fibers of the periodontal membrane are lost, and no new cementum is formed, connecting new fibers to the surface of the root, no functional stimuli are transmitted to the alveolar bone, and bone resorption sets in. Thus, the periodontal membrane widens (Fig. 3). Occasionally, resorption of the cementum is observed.

At any time, in the course of the development of the first stage in diffuse atrophy, repair is possible.

The widening of the periodontal membrane is not only the result of the loss of functional stimuli in certain bone areas, but there is a secondary factor: the increased pressure developing in the area of degeneration; due to edematous, hydropic swelling of the fibrous tissues and of the proliferation of capillaries. This increased pressure contributes to the resorption of the alveolar bone. That such increased pressure is active in the development of pathological conditions can be assumed, not only from the histopathological findings, but from the early clinical symptom of migration. This migration of the affected tooth always occurs in the direction from the diseased tooth surface.

As long as the second stage, i. e.: migration of the epithelial attachment does not set in and, providing that the systemic influences cease, regeneration may occur. Similar observations of regeneration are recorded in the comparative pathology of ligaments and tendons.

The question as to why degeneration in the periodontal membrane may involve only one tooth, or even a restricted area of one tooth, and, occasionally, all teeth, cannot be answered completely. Overstress of the periodontal fibers of one or more teeth is the explanation advocated by Box,[8] yet many facts speak against this assumption. The disease is often found in cases with a complete set of teeth in normal occlusion. In a dentition of this type, sometimes, only one tooth is affected, apparently, with no more stress than any of the other teeth. Moreover, the disease does not show any preference for pressure or tension side of the tooth. Orthodontic tooth movement produces pathological changes, similar to occlusal trauma. However, under normal systemic conditions, no lasting damage is done to the bone, or to the soft tissues.

Although trauma, as such, must be excluded as a causative factor in periodontosis, functional influence must be considered as a predisposing factor. From earlier investigations it is known[21] that teeth in occlusion are not in a resting state but in harmonious and balanced movement. There is a continuous, occlusal, active eruption, accompanied by mesial drift.[22] Part of this movement is compensated by attrition and balanced by occlusion. If all these processes take place evenly and smoothly the dentition is under ideal functional influences. If one of these components is disturbed the harmony is upset and the functional balance is thrown off. As long as the systemic condition of the individual is perfect, the functional stress of the periodontal membrane is compensated by rapid new formation of fibers and cementum. However, if there are toxins circulating in the system, or if the physiological metabolism is upset (endocrine dysfunction, nutritional deficiency, blood dyscrasias, toxins of micro-organisms, etc.) the reparative process is impaired and degeneration sets in. The presence of toxins in the system, or a disturbance of metabolism, may be primarily responsible for the disturbance of the balance in the dentition. The disturbance of functional adaptation, under these conditions, may explain why periodontosis is confined to one or more teeth, in one case, and why the entire dentition is involved, in another case.

Further explanation is required as to the finding of large epithelial rests in the perio-

dontal membrane, as illustrated in the bicuspid (Fig. 7). Similar epithelial structures were observed also in the periodontal membrane of other teeth in this dentition. These epithelial islands are quite different in their structure and size from those found normally. They are the remnants of Hertwig's epithelial sheath which, gradually disintegrates as the root forms and the tooth erupts. It is believed that, normally, the epithelial islands in the periodontal membrane are connected with each other when they are considered in three dimensions. In adults these epithelial remnants are in a resting stage. Their function is unknown, even questionable. Some investigators believe that the epithelial remnants may have an endocrine function. The assumption is that, in some way, the epithelial remnants may stimulate or activate the periodontal connective tissue in cementum formation.

The finding of enlarged epithelial rests, in this jaw with periodontosis, can be significant if Schour's observations[28] in hypophysectomized rats are taken into consideration. He found an increase in the number and size of the epithelial rests in the periodontal membrane of the incisor and molars of hypophysectomized rats. Schour's findings, and the finding of enlarged epithelial rests in the case of periodontosis, presented here, may be linked with the interpretation of clinical observations, suggesting a hypofunction of the pituitary gland as a probable causative factor in cases of periodontosis.[14] Tempting as such an interpretation may be, it is not conclusive and is submitted only as a possible explanation. It may apply to some cases, and not to others. It can be assumed that factors, other than hypophysectomy, might lead to similar hypertrophy of the periodontal epithelial rests as well.

Second Stage

The scarce clinical, as well as histo-pathological observations of the first stage, in the development of periodontosis, were explained by the fact that, shortly after the primary changes take place, these might be repaired or obscured by secondary pathological processes. The latter are migration of the epithelial attachment, along the surface of the root, and are the onset of an inflammatory reaction. A most interesting example of this stage was described in the publication of Gottlieb and Orban.[7] Some of the specimens will be reproduced and discussed here. Fig. 13, shows the interdental space between the right maxillary second and third molars of a case, at the age of twenty. In the apical region of the picture, a resorption can be observed which extends deep into the dentin of the second molar. The resorption has been partially repaired by cementum deposited on the resorbed dentin surface. The alveolar bone, in this region, is newly formed along the outline of the repaired resorption ("functional repair"). The epithelial attachment of this tooth extends from the bottom of the gingival crevice to the gingival margin of the resorption area, as seen in a higher magnification (Fig. 14). In this case, resorption of the root surface and proliferation of the epithelial attachment, along the root, took place simultaneously. This finding supports the previously quoted conception of Gottlieb that proliferation of the epithelial attachment, along the cementum, and resorption of the root surface, are the biological response of the epithelium and of the connective tissue, to certain changes in the vitality of the cemental surface. This was the first case to demonstrate an extensive proliferation of the epithelial attachment along the surface of the root, as occurring simultaneously with root resorption.

These findings help to explain other epithelial proliferations, as seen in Figs. 8 and 12. In the case, shown in Figs. 13 and 14, the epithelial attachment has migrated 3.2 mm. along the surface of the root of the second molar, extending only 0.88 mm. on the third molar. The bottom of the gingival crevice is at about the same level on both teeth. The epithelial attachment can be traced unbrokenly to the gingival border of the resorption area on the surface of the tooth. There is no space between the epithelium and the surface of the tooth, justifying the conclusion that the epithelium is firmly attached.

The migration of the epithelium, along the surface of the cementum, is possible only after the fibers of the suspension apparatus have lost their attachment to the cementum. As long as the fibers are attached to the surface of the tooth by continuously forming cementum, no epithelium can migrate along the root. Due to some toxic effect, degeneration of the connective tissue of the periodontal membrane is the primary pathological condition in the case of periodontosis, becoming apparent in the dissolution of the

Fig. 13—Interdental space between upper second and third molar of a case, 20 years of age.—Bottom of the gingival crevice at (a). The epithelial attachment (b), on the second molar, extends to the end (c) of a resorption area (d). The length of the attachment is 3.2 mm. The epithelial attachment of the third molar (e) is of normal length. The alveolar margin (f) of the third molar borders on a normal periodontal membrane, whereas the gingival part of the periodontal membrane of the second molar (g) is considerably broader.

principal fibers. Nevertheless, it is very probable that the highly differentiated cells of the periodontal membrane, the cementoblasts, are affected before any other changes take place. This condition would impair the formation of cementum. The only sign that continuous cementum apposition has ceased, is the lack of an uncalcified seam which, normally, forms the outermost layer of the cementum. Cessation of cementum formation, detachment, and degeneration of the periodontal fibers, allow the epithelium to migrate along the surface of the root. The migration of the epithelium continues until the end of the attachment reaches newly formed cementum where the fibers are attached.

In the specimen, showing the first stage in the development of periodontosis, an epithelial proliferation can be observed in the area of the widened periodontal membrane (Figs. 3 and 8). This proliferated epithelial structure adjoins the cementum. None of the sections of this specimen shows a connection of this epithelium with the marginal epithelial attachment. The presence of an isolated epithelial attachment, separated from the marginal gingiva (Fig. 8), is a unique observation. There are different explanations for the presence of this isolated epithelial attachment to the cementum. It may be that, due to a toxic influence in this area, an epithelial rest of the periodontal membrane attached itself to the cementum after cementum formation had stopped. No newly formed, uncalcified cementum can be seen between the cementum and the epithelium. The same explanation, which was given for the migration of the epithelial attachment along the root surface, may apply

Fig. 14—Higher magnification of the area (c) in Fig. 13; (a)—epithelial attachment which ends (b) at the beginning of a resorption area (c); (d)—bony repair on the base of the resorption area.

to the attachment of an epithelial rest to the root surface.

It is not impossible that this epithelial island has a connection with the marginal epithelial attachment which could not be detected in the available sections. This assumption is the most plausible for the conditions shown in Figs. 10, 11 and 12. Here also is an isolated epithelial attachment on the mesial side of the second bicuspid. This epithelium is separated from the marginal epithelial attachment by strong fiber bundles. However, it may also be the extension of an epithelial attachment from another side of the tooth. Below the end of this seemingly isolated epithelial proliferation there are fiber bundles, attached to the root surface by newly formed cementum. The findings, shown in Figs. 8, 11, 12, 13, and 14, indicate that the proliferation of the epithelial attachment in periodontosis may develop as a continuous extension of the marginal attachment; or by a confluence of the marginal epithelial attachment with the proliferated attached epithelial rests of the periodontal membrane.

The occasional observation of root resorption, as a symptom of periodontosis, can be explained also by the same factors which cause the migration of the epithelial attachment. The continuous apposition of cementum is the prerequisite for an undisturbed function of the teeth.[24] Should the new formation of cementum stop, because of some toxic effect upon the cementoblasts, the root surface will not be covered by uncalcified cementum, as it is under normal conditions, but will calcify entirely. This calcified root surface seems to be more susceptible to resorption than that covered by a newly formed, uncalcified layer, as has been demonstrated by previous investigations.[25, 26]

The damage, done by root resorption, can be repaired if the cause is eliminated[2, 4] but, repair of the damage, done by proliferation of the epithelial attachment, is doubtful.

In most cases, where the epithelial attachment shows extensive migration, a chronic inflammatory reaction is present. This inflammatory reaction is due probably to toxic influences, originating in the gingival sulcus.

Third Stage

As previously discussed, the clinical picture of the first stage, in the development of periodontosis, is characterized by migration of the tooth without pocket formation. The second stage cannot be clinically distinguished from the first, the only characteristic feature of this stage being proliferation and migration of the epithelial attachment. The sole clinical difference between the first and second stage is that the pathological condition may become entirely repaired in the first stage, whereas, after epithelial proliferation in the second stage, complete regeneration is impossible. Both stages are of relatively short duration. The proliferated epithelium may not stay attached to the root surface for a long period. It may be separated to such an extent that it forms a deep pocket. It is a known fact that the gingival sulcus is a potential trap for accumulating deposits from the saliva. Here, micro-organisms find a good medium for their growth. The toxins, produced by these organisms, penetrate into the epithelium and underlying connective tissue. Inflammation sets in, in the connective tissue and the epithelial cells, which line the pocket, degenerate. This constant irritation and degeneration of the cells of the epithelial attachment, at the bottom of the sulcus, can be considered as the causative factor in the separation of the epithelial attachment from the tooth at this location. The toxins may penetrate between the intercellular spaces of the epithelium and along the root surface, thus weakening the attachment, and preparing for separation. Toxins may originate also in the inflamed connective tissue, behind the epithelial attachment, where the inflammatory reaction leads to toxin production. In this way, the sulcus becomes increasingly deeper, more deposits are lodged, more bacteria accumulate, and the inflammatory reaction becomes increasingly severe. The inflammation in this third stage must be considered a *secondary* pathological condition. The inflammation will progress in the same way as inflammatory reactions in cases of primary gingivitis; that is, along the pericapillary lymph spaces into the bone marrow, producing an osteitis.[10] This process will lead to progressive bone resorption, gradually destroying the supporting tissues.

In the third stage of periodontosis, the most characteristic feature is the development of the deep pocket, or pockets, with all the symptoms of inflammation. The inflammation aggravates the primary pathological condition, causing more rapid bone resorption, migration of the epithelium, along the

surface of the root, and separation of the epithelium from the cementum. In the course of these events, remissions and relapses may occur. If a deep pocket becomes obliterated, acute inflammation may set in and abscesses or retention cysts develop, as is shown in Fig. 10. Gottlieb claimed that the separation of the epithelium from the surface of the tooth is a biological reaction of the epithelium to the tooth surface, and that this severance is independent of external influences. The morphological result of this reaction is the formation of the hornified cuticle. Under normal conditions, this biological reaction may play the most important part. In pathological pocket formation the inflammatory reactions obscure and aggravate conditions.

It is apparent that teeth, affected by these processes, lose much of their supporting tissues. Under such conditions even teeth in normal occlusion may show signs of secondary traumatic changes in the periodontal membrane.

SUMMARY

In the development of diffuse atrophy, or periodontosis, three stages were differentiated: The *first stage* is characterized by degeneration and desmolysis of the principal fibers of the periodontal membrane. The first consequence of this process is probably the cessation of cementum formation. Simultaneously, resorption of the alveolar bone sets in, due to lack of functional stimuli from the tooth, as well as an increase in tissue pressure, resulting from the accumulation of edema and the proliferation of capillaries. This increase of tissue pressure also accounts for the migration of the tooth from the affected area. Intrinsic factors have to be considered primarily, as a cause for the initiation of pathological events which bring about a complete picture of periodontosis. Such factors may be found in the dysfunction of different endocrine glands, toxins from different sources, metabolic disturbances, dietary deficiencies, etc. The preference of the disease for a certain localization may be due to a functional insufficiency of the suspensory apparatus. This functional insufficiency can be attributed to toxic influences which impair the physiological regenerative process in the connective tissue of the supporting structures, exposed to constant mechanical influences.

The *second stage* is characterized by the rapid migration of the epithelial attachment along the affected cementum. There may also be a proliferation of the epithelial remnants in the periodontal membrane. With the epithelial proliferations the first signs of an inflammatory reaction become evident. The inflammation can be explained satisfactorily by the penetration of toxins from the gingival crevice. These toxins follow the course of capillaries as well as epithelial structures. The second stage can not be distinguished, clinically, from the first. Both stages are of short duration. They may differ in their consequences; the first stage may be completely repaired, if the primary causes cease; whereas, the second must lead to the third stage.

The degeneration of the epithelial tissue, at the bottom of the gingival crevice, causes the development of deep pockets and, thus, the *third stage* follows in the development of periodontosis. The inflammatory reaction is aggravated, leading to an obliteration of the primary symptoms. Large portions of the supporting tissues are destroyed and even normal occlusal forces cause secondary traumatic changes. This third stage is clinically most frequently observed.

The knowledge of the sequence of the events in the development of periodontosis is the prerequisite for wise treatment.

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Announcement

THE PERIODONTIA DEPARTMENT of New York University College of Dentistry cordially invites each investigator in the field of periodontal disease who has published a paper or papers containing the results of either laboratory research or clinical observations which bear directly or indirectly upon the etiology or treatment of periodontal disease in man or animals (1) to submit a concise summary of those of his papers which he deems to be his basic contributions to this field and which he considers to be in agreement with his present thinking, or with his experience and thinking up to a time specified by him; and (2) to cite the location and time of appearance of such paper or papers which contain such basic findings; also (3) to enclose any additional statement that may seem necessary to him to amend the foregoing statements so that they more accurately reflect his present opinions.

These summaries will, therefore, contain only present opinions, rather than the total results of each worker's endeavors which may, in addition, include errors, duplications, altering viewpoints, etc. The compilations of such summaries which will *contain the essence of each investigator's present thinking will then be published in* BOOK FORM and will, therefore, not be a series of abstracts of papers on either the etiology or treatment of periodontal disease.

The summary should be in our hands not later than March 15, 1942. (209 E. 23 St., New York, N. Y.)

Editorials

Here is Your Hat . . .

THE bromidic title of this editorial reminds us of the man of the house, who having given up hope of some late callers ever leaving, said to his wife, "Well, Abigail, we had better retire so that our guests can go home."

In recent months some well meaning hosts within the dental profession have been considering, quite seriously, giving us our hats, together with other small organized national specialty groups in dentistry. This is all the more astonishing because the "Windy City," the most recent source of such intentions, is in the mind of most dentists the last word in hospitality.

The Chicago Dental Society and some members of the American Dental Association believe that the small specialty groups are detracting and interfering with the meetings of these larger organizations, although the small groups usually hold their meetings preceding those of the major societies.

Inasmuch as the owner of this Journal, the American Academy of Periodontology, is one of the small groups under consideration, it is a timely topic for discussion and one point of view is here presented.

The relative size of this group is of interest. The membership of the Academy is less than two hundred. Compare this figure with the more than 50,000 members of the A. D. A. and the large membership of the Chicago Dental Society with its thousands of loyal adherents!

The American Academy of Periodontology was organized *twenty-eight* years ago and did not at first meet in the city selected by the American Dental Association for its annual meetings, but in another locality. Much dissatisfaction was then expressed by members and non-members because they felt the meetings of the Academy should be available to those attending the A. D. A. Many of the Academy members were active in the parent society and could not afford either the time or expense to attend both meetings unless they were held in the same city at about the same time. The change in place of meeting was therefore made and the meetings held the last three days of the week preceding the meetings of the A. D. A. Some of the Academy members are trustees and delegates of the mother society, one was a recent president, and each year some are section officers, essayists, and clinicians.

At the formal and urgent request of the American Academy of Periodontology, periodontia was given a section in the A. D. A. first, as a part of the Orthodontia section only, and later, again by request, a full section was allowed. It is worth noting here that it is always one of the best attended sections. It has been sponsored by the Academy since its beginning.

There are many members who arrive at the periodontists' meeting one or two days late so as to be able to attend more of the A. D. A. meeting; it is necessary to wait over two days before its sessions start.—A proof of loyalty if one is needed.

The Academy meetings are open to all A. D. A. members; this small society is well represented in the activities of the parent society; it is vitally concerned with

all matters relating to dentistry; it has been in existence for twenty-eight years during which time the A. D. A. has nearly doubled in membership; therefore this specialty group, we are convinced, does not detract from the interest or attendance of the annual meetings of the A. D. A.

Past-president Robinson of the American Dental Association recommended in his report to the Board of Trustees at Houston that a committee be appointed to study this subject of the trend of the specialty groups to "follow a path of thoughtless secession from the parent body," and to hold their meetings independent of it as to time and place.

The Chicago Dental Society has before it some *Resolutions* which call attention to the number of small organizations who hold meetings, business and literary, at the time of their Midwinter Meeting, and discourage all such arrangements.

The national auxiliary groups meeting in Chicago at this time are mostly executive boards, committees, and officers of these societies, who need to get together during the year to facilitate their work. The number in attendance is so small that we question any interference with the Chicago society; we believe rather that it increases the attendance.

Three Score Years and Ten

WE RECALL, with gratitude, the men who have contributed to the development and advancement of dentistry of the past and present; we confess that we are indebted to them all, permanently. However, we welcome an opportunity at this time to pay our respect to one in particular who stands out among many of these men as having won a real place in our esteem. His devotion to dentistry and his contributions to its advancement in the fields of education, scientific research, and journalism are too familiar to need repetition here.

We therefore salute Dr. William J. Gies who had faith in the soul of dentistry; who stimulated our self respect as dentists; who organized opportunities and furnished inspiration for the continual progress of our profession. On February 21, 1942, Dr. Gies celebrates his seventieth birthday. Our wish for at least twenty more birthdays for him is inspired by the lines of Dryden:

"Fate seemed to wind him up for four score years;
Yet freshly ran he on ten winters more."

Dental Abstracts

DURING this year, the American Association of Dental Editors is trying out a well formulated plan for obtaining abstracts of papers, published in current dental literature, from the authors themselves. It is believed that they are best qualified to write informative abstracts of their own papers. A similar method has been used successfully by *Biological Abstracts*.

The *Journal of the American Dental Association* will publish the 1941 abstracts procured by the editors' organization.

The successful result of the plan will depend upon the coöperation of writers who contribute to our dental periodicals and to the programs of our dental meetings.

—G. R. S.

ABSTRACTS AND EXTRACTS OF CURRENT DENTAL LITERATURE FOR 1940 (Continued from 12(2) July, 1941)

Symptomatology and Diagnosis, Treatment

(e) Symptomatology and Diagnosis

(Continued from 12(1):34. Jan., 1941)

340—Austin, L. T.: **Relationship of dental sepsis to systemic disease.** *J. A. D. A.* **27**:684-688. May, 1940.

Dr. Austin has obtained the opinions of consultants in various sections of the Mayo Clinic regarding the relationship of dental sepsis to their particular fields of systemic disease. Their opinions follow:

Dr. Eusterman thinks that foci of infection in the mouth can cause gastritis and acute and subacute ulcers of the stomach and duodenum. Dr. Bargen states that the streptococcal type of ulcerative colitis has been proved to be related to dental infection. Dr. Willius feels that periapical infection may be the starting point for subacute bacterial endocarditis, but there is not yet any convincing evidence. Dr. Benedict feels that foci of infection in the mouth may be the cause of some types of eye disease, especially of the iris and choroid. Dr. Slocumb states that removal of infected foci, including infected teeth, is justified in all patients with infectious arthritis; and if the arthritis is difficult to control, the removal of questionable foci, such as devitalized teeth and dental roots, should be advised. Removing infected teeth, cleaning up the mouth, and removing poor crowns is especially important in syphilis, Dr. O'Leary believes, since infection in the mouth is a factor in salivation from mercury and deposits of bismuth, in syphilis therapy. Dr. Thompson says that the relationship of infections in the genitourinary tract to focal infection elsewhere in the body, especially infected teeth, is definite. Dr. Rosenow states that dental foci of infection are undoubtedly a common cause of systemic disease; but the need for further consideration of the problem is apparent.

—*Martin Blumenfeld*

341—Bacchi, J. G. and La Rocca, V.: **Relation between corneal ulcers and diseases of the mouth.** *D. Digest.* **46**:15. Jan., 1940.

The authors present a case which to them, proves the theory of selective localization. The patient had ulcers of the cornea of both eyes, with lacrimation, violent pain, and photophobia. Since childhood, he had been affected with trachoma, for which he had been treated.

In addition to usual treatment for ulcer and secondary iritis, a peritomy was performed on both eyes; foreign protein therapy and hemotherapy were also employed. A few weeks of this treatment showed no result.

The patient was then sent to the dentist for a complete oral examination. The gingiva was found to be inflamed, and receded; radiograms showed typical periodontitis with pathological bone absorption of horizontal type. A bacteriological examination showed the same organisms as in corneal ulcers. Within one month, all the teeth were extracted. After the initial period of exacerbation, the symptoms subsided and the condition cleared up. This proved that the eye infection was secondary to the focus of infection in the mouth, with dissemination through the blood stream.

—*M. B.*

342—Bober, H.: **Beitrag zur Frage der Bedeutung der Paradentopathien bei Schulkindern und Jugendlichen fuer die Gesundheitsfuehrung.** Contribution to the question of the importance of paradentopathy, in school children and young people, to the general health. *D. Z. W.* **3**:40. 1940.

The most important point in this problem is the conception of the dental organ as a biological whole, which does not have independent existence in the organism, but is united with the general metabolic processes of the organism.

Studies were made on Berlin school children, considering: the quality of the gingivae; adherence of the gum to the tooth; swelling of the interdental papillae and gingival margins; ease with which the gums bleed; occurrence of vestibular and lingual gingival pockets, occurrence of supra- and sub-gingival calculus. The article is well and fully illustrated with charts and tables.

Results of these studies:

1. Paradentopathies in school children must be considered a stigma of constitutional-biological eccentricity.

2. 100% of children studied from a constitutional-anthropological viewpoint showed capillary disturbances.

3. 100% of children showed severe disturbance of electrolytic equilibrium of their bio-colloidal construction.

4. Differences in paradental conditions were related to social environment.

5. Differences in paradental conditions were related to living conditions.

6. Existing paradentopathy should be corrected, to prevent more serious developments.

—*M. B.*

343—Brauer, R.: **Zahnfleischerkrankungen in der Schwangerschaft.** Diseases of the gingivae in pregnancy. *Med. Klin.* **35**:280-82. May 3, 1939.

Gingivitis gravidarum occurs, clinically, about the third month of pregnancy as a diffuse edematous inflammation of the gingival margins, which become livid, and bleed easily on pressure. All these symptoms usually disappear right after parturition. Dr. Brauer discusses the various ideas which have been presented on the etiology of this condition. He considers it a symptom of disturbance of the general metabolism, and thinks that it requires no external treatment, in a healthy mouth, assuming that any possible vitamin deficit has been recognized. However, in cases of gingivitis, where exogenous factors (overhanging fillings, calculus, etc.) are present, local treatment is, of course, necessary. The mouth should be kept as sanitary as possible, during pregnancy, to prevent damage to the dentition and the results of gingivitis gravidarum.

—*M. B.*

344—Goldman, Henry H.: **Acute aleucemic leucemia** (Case Report). *Am. J. Orth. & Oral Surg.* **26**(1):89-92. Jan., 1940.

There are several forms of leucemia, myelogenous, lymphatic and monocytic, to which Goldman adds acute leucemia. This case of acute aleucemic leucemia illustrates the effect of systemic disease on oral tissues. Patient complained of oral symptoms and was treated for Vincent's infection with salvarsan, without improvement. Systemic symptoms were almost entirely absent at first presentation, but became progressively worse, with fatal termination two days later. Detailed blood picture is given; this and the condition of bone marrow were pathognomonic of acute aleucemic leucemia. Oral mucosa was infected and slightly swollen. Minute petechial hemorrhages were scattered throughout the mouth; gingivae were hyperemic and necrotic, of a dark blue color. Tongue was slightly swollen and coated with grayish white material; breath foul; teeth very loose. Microscope revealed marked gingivitis with complete absence of epithelium. The dense connective tissue of the gingival corium was replaced by loose strands with numerous blood vessels, and the entire structure was studded with myocytes. The transseptal fibers had almost entirely disappeared with only occasional remnants still discernible.

—*H. M. Goldman*

345—Goth, A.: **Eine Methode zum Nachweis der C-Hypovitaminose.** A method for the determination of hypovitaminosis C. *Z. Vitaminforschung.* **10**: 15-25. 1940.

Hypovitaminosis C can be determined by a test of the urine or blood. Urine analysis is the most commonly used method today; in this, vitamin C is given to an individual daily until at least one half of the given dose appears in a 24 hour sample of urine. By daily intravenous injection of 300 mg. vitamin C this occurs in 1-5 days, when the organism is saturated; in hypovitaminosis, it takes 6-10 days or more. For the titration, 2,6-dichlorphenolindophenol is used.

In the case of blood analysis, a value of over 1 mg. % of ascorbic acid indicates saturation; a value of less than 0.4 mg. % shows hypovitaminosis. Values of 0.4-1.0

mg. % allow no conclusions as to the vitamin C state of the organism. In such cases, 300 mg. of ascorbic acid must be given intravenously, and another determination made in two hours. If there is a rise of over 0.5 mg. %, the organism is saturated; no rise, or only a 0.3—0.4 mg. % increase, indicates hypovitaminosis. Using this method, the author worked on patients with various conditions and diseases, and has presented tables of the results of his study.

—*M. B.*

346—Hofer, O., and Pini, C.: **Das Verhalten des weissen Blutbildes bei der Alveolarpyorrhoe.** The conditions of white corpuscles in alveolar pyorrhea. *Z. Stomat.* **38**:77. Jan. 26, 1940.

Assuming that alveolar pyorrhea is a chronic disease in which septic symptoms can develop, and in which white blood corpuscles may be affected, the authors began their studies in a fashion analagous to studies on the effect of infected tonsils on white blood cells. Through thorough massage of gingival pockets, it was thought to obtain resorption of the contents of the pockets, and, at the same time, to prevent resorption of these contents by way of the alimentary tract and so give prominence to the action of parenteral assimilation. As control, the same studies were made on people who showed no signs of pyorrhea. Blood samples were taken by pricking the finger; differentiation of the elements was done in a Thoma-Zeiss counting cell. In evaluating the blood picture, the authors used Arneth's formula; shifting of the white cells to the left serves as an indication of the existence of an infectious process.

It was shown that in pyorrhea, massage of the pockets leads to a considerable and immediate rise in the white cell count, which, because of the arrangement of the experiment used, must be traced back to the toxic products in the gingival pockets; this continual attack of the toxic products on the hemopoietic system cannot be without effect on its ability to react.

—*M. B.*

347—Holdren, H. T.: **Periodontoclasia and the Dunlop oxygen treatment.** *D. Items Int.* **62**:134-137. 1940.

Periodontoclasia presents a complex group of symptoms; these are discussed under three headings:

1. Virulent type (acute symptoms) early shows marked local symptoms. The gingival tissue becomes swollen, red, and sore, and bleeds on the slightest irritation. Easily distinguished pockets then form in the bony process; fine granular deposits may be found on the peridental membrane, which may become larger and tenacious.

2. Non-virulent type (chronic symptoms) presents no distinctive symptoms to betray the true character of the local conditions. The gingiva shows no marked changes; pockets are found in the bony process, with less tendency to lateral extension. The peridental membrane is not so much affected; pus does not appear on the surface.

3. Catarrhal type is more chronic and atrophic; the soft tissue is shrunken and anemic, and the bone is markedly atrophied; there is a great deal of malodorous secretion. The radiograph is a great aid in making the diagnosis and prognosis.

Dr. Dunlop developed his oxygen treatment after finding vaccines ineffective. The dry vapor (a non-irritating oxygen gas) is forced into the mouths of the main ducts; this produces a leucocytic infiltration, following a preliminary hyperemia. When this condition is obtained, the vapor may be introduced around all the teeth; this forces the organisms directly into the wall of leucocytes, where they are destroyed; their toxins are liberated and distributed throughout the body. This oxygen treatment is thus a form of auto-vaccination.

—*M. B.*

348—Lempke, H.: **Ein Beitrag zur Frage des Zusammenhanges zwischen Zahnfleischentzuendungen und Allgemeinserkrankungen.** Contribution to the question of the relationship between gum inflammation and general disease. *Z. Rund.* **3**:63. 1940.

The author proceeds from the viewpoint of the human organism in its entirety, and shows how this view has affected the whole of dentistry. The relationship between inflammations of the gingivae and disturbances of normal function of the blood-forming organs, as well as with poisoning by mercury, bismuth, and especially lead compounds, is discussed; the connection with paradentosis is closely examined. The author

further studies the conditions in disturbances of vitamin metabolism, and shows the etiological relationship between diseases of the oral mucosa and the so-called vitamin deficiency diseases. From these facts on the relationship of general diseases to stomatitides, conclusions are drawn as to therapy. A critical survey of special treatment of inflammations of the gum underscores the necessity for an adaptation of therapy to biological exigencies, through a study of natural resistance of the organism. The experiences of Heisler, with coffee-charcoal as a therapeutic agent, are briefly mentioned. In conclusion, the difficulties of constructing a schematic plan of cure are shown.

—*M. B.*

349—Miller, E. L.: **Pericoronal infection.** *D. Student's Mag.* Feb., 1940.

The most frequent offender is the mandibular third molar, although any of the posterior teeth could be thus involved. This is due to the prevalence of crowding of the lower arch, thus preventing full eruption of the lower third molars. Pericoronitis may be either acute or chronic. In the chronic type, there is little or no pain, and almost complete lack of swelling or soreness of the surrounding parts; it may affect a completely unerupted tooth as an area of bone rarefaction. The acute type occurs only in partly erupted teeth. It may be traumatized by the upper third molar. The bacteria found under the flaps are the common inhabitants of the mouth; it may become the incubation zone for Vincent's infection.

The local symptoms of pericoronitis are red, inflamed, hypertrophied flap of tissue, surrounding the crown of the erupting tooth; swelling may be present both at the site and in cervical lymph nodes. There may be difficulty in swallowing, due to involvement of the superior constrictor muscle of the pharynx. The possibility of infection from pericoronitis being transported to another part of the body is not at all remote.

In the treatment, a decision must first be made as to whether the tooth is to be removed or retained. Opinion as to whether or not to treat before extraction, if extraction is indicated, is somewhat controversial. Whether the flap is excised or the tooth is removed, infiltration anesthesia is contraindicated; either local conduction or general anesthesia should be used.

—*M. B.*

350—Pelzer, R. H.: **A method of plasma phosphatase determination for differentiation of alveolar crest bone types in periodontal disease.** *J. D. Res.* **19**(1): 73-85. 1940.

The purpose of this investigation was twofold:

1. To trace such changes in phosphatase value as occur both locally and in the region of various types of alveolar bone decalcification, and in the blood plasma from the finger;

2. To evaluate the validity of such phosphatase values as an index of the rapidity of alveolar bone loss in comparison to radiographic findings.

Radiographic findings in periodontal disease show three types of bone destruction: arrested, slow, and rapid. Plasma phosphatase is markedly increased in certain generalized bone diseases; Labbé has shown increased phosphatase, also, in alveolar bone of fresh cadavers where evidence of bone destruction was present. All methods of phosphatase estimation are based on its action in vitro of hydrolyzing phosphoric esters to free an increased quantity of inorganic phosphate; for this work, the micromethod of Lundsteen and Vermehren was used.

On comparing phosphatase values from gingival blood with that from finger blood, a significant variation toward a greater concentration of phosphatase in the plasma from the region of radiographically indicated alveolar bone crest dissolution, was found. In cases of normal alveolar bone crests, plasma phosphatase concentration in this region tends to be closely similar to values found for individual normal as determined by plasma phosphatase of finger blood.

—*M. B.*

351—Rebel, H. H.: **Gingivitis und Stomatitis. Nichtspezifische entzuendliche Erkrankungen des Zahnfleisches und der Mundschleimhaut.** Gingivitis and stomatitis: non-specific inflammatory diseases of the gingivae and oral mucosa. *Dtsch. Zahn, Mund, und Kieferhik.* **7**(4):220-38. 1940.

Dr. Rebel presents a discussion of the histology of the oral mucosa, and something of the anatomy of gingival pockets. He considers that both local tissue conditions and the factor of inflammatory conditions are involved in producing the clinical picture of stomatitis and gingivitis; inflamma-

tory conditions can be of the acute type (catarrhal, ulcerous, or necrotic-gangrenous), or of the chronic type (exudative and alterative, with atrophy or hypertrophy). He presents a schema of the known diseases of the gingiva, using three classifications: simple catarrhal form (gingivitis simplex), simple catarrhal with hypertrophic character, and ulcerous form (gingivitis ulcerosa); the clinical picture and therapy for each type are given. He also presents a schema for the course of therapy.

The author then discusses the factors, other than local, which are involved in these diseases, including climate and environment, Vincent's infection, metal poisoning, vitamin deficiencies, blood diseases, and conditions in which profuse bleeding occurs.

The article is a comprehensive survey of gingivitis and stomatitis, and it is difficult to present a resume of it because of the wealth of material. The author brings out both the local character of the conditions, and their close association with general body diseases, of which they may be an early or a late symptom. Their therapy involves a knowledge of the whole field of medicine.

—*M. B.*

352—Shapiro, B. B.: **The oral lesions and blood picture of pernicious anemia, leukemia, and agranulocytosis.** *D. Items Int.* **62**:113-126. Feb., 1940.

There is first a short introduction of the work of various men in correlating oral lesions with blood diseases. Then follows a picture of the normal blood giving a blood count and differential. Taking each disease separately, the author presents the etiology, oral pathology, and blood picture; he has drawn from various authorities and quotes directly.

In pernicious anemia, Hunter's tongue and stomatitis are dependable oral symptoms. The tongue is devoid of white coating, is smooth and round at the edges, with papillary atrophy; it may be ulcerated. The oral mucosa is usually pale. The various types of leukemia present similar oral lesions. The gums become swollen and tender, painful, and dark chocolate in color, the teeth become loose and movable, petechial hemorrhages may appear in the oral mucosa, and the patient usually has a foul mouth odor.

Agranulocytosis in its early manifestations is confined solely to the mucosa of the mouth, throat, and tonsils; it may simulate Vincent's infection. The outstanding symptoms are sore mouth, with swelling and reddening of the gums; shallow ulcerations and indurations of the gums, lips, tongue and buccal mucosa, associated with a moderate degree of regional adenitis. From these locations, Vincent's organisms can be obtained in large numbers.

Extensive bibliography.

—*M. B.*

353—Winter, L.: **Blood dyscrasias: their oral manifestations.** *Am. J. Orthodont. and Oral Surg.* **26**:67-79. Jan., 1940.

Dr. Winter reviews those blood dyscrasias which show some mouth lesions:

Primary pernicious anemia: The principal oral manifestations are really pathognomonic symptoms of this disease. Moeller's glossitis or Hunter's tongue is present in 75% of cases; the oral mucosa is usually pale.

Chlorosis: The oral mucosa is pale, gingivitis and periodontoclasia are present; a great number of cavities occur and there is softening of the dentin.

Secondary anemia: Pallor of the lips, tongue, and mucosa; gingivitis present with marginal bleeding.

Polycythemia: Bleeding gums; the skin and mucosa are cyanosed and purplish-red.

Leucemia: There is hyperplasia of the lymphatic structures of the tongue, pharynx, and tonsils; nodular swellings of the gums appear, and the gums do not bleed easily. Later, gangrenous stomatitis and large sloughing areas may be present.

Scurvy: There is marked stomatitis; inflammation of the mucosa, gums are spongy, swollen, and bleed easily; marked fetid odor. The periosteum and periodontium are involved, and the teeth become loose.

Purpura: Petechiae and hemorrhages occur in the gums and mucosa.

Agranulocytosis: Ulcerative and necrotic lesions, mainly in the posterior portions, and usually bilateral. (It must be distinguished from Vincent's infection and pericoronitis.)

—*M. B.*

(f) Treatment

(Continued from 12(1):53. Jan., 1941)

590—Ahrens, H.: **Paradentose und Zahnarzt.** Paradentosis and the dentist. *Dtsch. Zahn. Wschr.* **43**(12):192-94. 1940.

This article is written in the form of an open letter to Dr. Fuchs, referring to his article in a previous issue of the same journal. The author respectfully contradicts Dr. Fuchs, who stated that paradentosis can be cured by proper diet, especially an adequate vitamin C intake. Dr. Ahrens contends that, as regards the dentist's relation to paradentosis, it is no longer a problem, but a conception; through knowledge of its pathogenesis, the dentist has received stimuli in the entire field of dentistry, so that it can be said that paradentosis is the key to all of it (dentistry). The author supports the viewpoint of periodontists who use surgery as *one* means of treating paradentosis, and who feel that not every case can be cured by internal therapy and vitamin administration. He also feels that malocclusion, in its various phases, is a very important etiologic factor in paradentosis, and, of course, *this* cannot be influenced by internal therapy. The role of the dentist is to conceive of paradentosis in all its bio-mechanical aspects, and treat all the various causative factors in each case.

—*M. B.*

591—Armentano, L.: **Der Vitamin C-Bedarf und dessen Deckung.** Vitamin C requirement and means of meeting it. *Z. Vitaminforschung.* **10**:6-15. 1940.

A state of saturation of vitamin C is regarded as an optimal condition. To determine the daily requirement, it is necessary to make a saturation determination twice on the same individual; this daily requirement is then calculated from figures for saturation at both times and the amount needed for them. In this way, it was determined that the daily requirement is 45-56 mg. In cases of fever, infection, in sports, and heavy manual labor, the need is considerably higher.

Preserves of Hungarian paprika (which were tested in this experiment) do not lose any of their vitamin C value, in spite of the manner of preserving.

Using the preparation "Pritamin," 10 gm. a day suffice to cover the daily requirement; and giving 50 gm. daily, the deficiency existing in severe infections can be overcome in 9-10, or at most 14, days. In experimental cases, where 300-500 mg. crystalline ascorbic acid were given daily for 4-6 weeks, no symptoms of hypervitaminosis C were observed.

—*M. B.*

592—Blanquie, R. H.: **A consideration of the surgical treatment of periodontal lesions.** *J. A. D. A.* **27**:757-762. May, 1940.

This article deals with the surgical procedure involving complete eradication of all disorganized and diseased soft tissue forming and underlying the pocket, bringing the line of incision entirely within healthy, normal structures. This therapy does not require the removal of any portion of the alveolar process, unless warranted by certain conditions which occur but occasionally. Prior to operating, it is advisable to medicate the periodontal pockets with a 5% solution of mercurochrome. A description of the actual operating procedure is given in detail. An essential part is protection of the areas operated upon by the use of suitable surgical dressing; the principal ingredients of which are zinc oxide and rosin, in the powder, and oil of cloves and olive oil, in the liquid. If excessive bleeding is a possibility, tannic acid powder may be added to the mixture. Upon removal of the packing, the granulation tissue should be stimulated by proper brushing.

The surgical method of treating periodontal lesions offers the most expeditious means of therapy. It completely eliminates the periodontal pocket, which is replaced by healthy tissues and a gingival crevice of normal depth; it also insures the thorough eradication of all pathogenic processes that make up the disease entity, periodontoclasia, and thereby removes from the oral cavity a serious menace to health.

—*M. B.*

593—Box, H. K.: **Case management of simplex and complex periodontitis.** *D. Items Int.* **62**:915-921. Oct., 1940.

In simplex periodontitis, gingival inflammation dominates the clinical picture, with

fairly even distribution of sites; increased tooth mobility is a late symptom; pocket formation is usually early but slow. In complex periodontitis, increased tooth mobility is an early sign, with uneven distribution of sites; gingival inflammation is inconspicuous; pocket formation is late but rapid. Vitamin C deficiency is an important factor in producing the clinical picture; Sinclair, however, maintains that there is a polyavitaminosis. Dr. Ham states that "in long continued slight vitamin C deficiency, bone which normally wears out is only meagerly replaced; in due time, it becomes unable to bear its load, and obvious lesions appear."

The normal gingival tissue appearance is described, with an explanation of "stippling"; gingival edema and McCall's festoons are also described. Kronfeld has stated that actual tissue examination does not substantiate the supposition that loss of bone and pocket formation must go together. Therefore, pocket depth must be determined clinically. The use of the measuring probe is a simple method; however, the injection of a radiopaque substance followed by radiography is much more accurate.

—*M. B.*

594—Brodsky, R. H.: **Grenz ray therapy in periodontoclasia and oral tuberculous lesions.** *D. Items Int.* **62**:927-934. Oct., 1940.

The Grenz ray is a series of electromagnetic oscillations having a wave length of approximately two Angström units; it permits a diversification of dosage in the individual strata of the tissues, making possible the absorption of the ray where it is most desired without penetrating the deeper structures. The author reports very good results in the use of the Grenz ray in oral tuberculous lesions, pain usually disappearing in 24 to 48 hours. This led him to believe that it might be effective in treating certain types of periodontal lesions, since here, too, we are dealing with localized inflammatory processes.

Dr. Brodsky then discusses briefly the various types of gingival diseases, and periodontoclasia, with their causes. Some patients received only Grenz radiations; others, scalings and subgingival medications; and still others, combinations of both the radiations and the subgingival therapies. He found from the very beginning that the radiations were having very definite beneficial effects on the gingival mucosa. In cases of extensive advanced chronic suppurative forms of periodontoclasia, the dosage had to be greatly increased. With high dosages, the suppuration was greatly reduced or entirely eliminated. Concluding, the author feels that Grenz radiation is an important adjunct to the accepted methods for the treatment of periodontal lesions.

—*M. B.*

595—Brosch: **Ein Beitrag zur Kenntnis der Ergebnisse der Radikaloperation bei apikaler Paradentitis.** A contribution to the knowledge of the results of radical operation for apical paradentitis. *Z. Rund.* **49**(10):348. 1940.

Radical operation is superior to other methods of therapy for apical paradentitis, in that it can cure cases which offer stubborn resistance to conservative treatment. Regeneration of bone, destroyed by infection, always follows. Weakening of the supporting mechanism of the tooth is proportional to the surface of the amputated root, with an intact paradental membrane. An overburden produces further weakening through exhaustion of the paradental tissues, which can, however, be equalized by hypertrophy of the other tissues. There is found no functional band of paradental fibers from the remaining section of the root to the newly regenerated alveolar process. The whole article is illustrated with good photomicrographs.

—*M. B.*

596—Dement, R. L: **The flap operation and its place in the treatment of periodontal lesions.** *J. A. D. A.* **27**: 1761-1764. Nov., 1940.

In the treatment of suppurative periodontoclasia, there are four favored technics in use at this time: subgingival curettage (conservative treatment), flap operation (full, and modified), gingivectomy, and electrocoagulation. Much depends on the skill of the operator and the particular case in hand, as to which method to use. In deep lesions, where there is some doubt as to whether subgingival curettage can permanently relieve the pathologic condition, the flap operation is very satisfactory, since it conserves the overlying gingivae and, with

the blood clot acting as a scaffolding, there may be a regeneration of the alveolar process. In the anterior part of the mouth, the flap operation is the procedure of choice; with posteriors, it is also good procedure, but slightly more difficult.

The full-flap operation consists of two incisions parallel to the long axis of the teeth, starting at the gingival border in the interproximal spaces, and extending apically to the region of the apex; another incision is made in the interproximal area, mesiodistally, separating the buccal or labial gingiva from the lingual. The modified flap operation necessitates only one incision, mesiodistally in the interproximal mucoperiosteum, and as near the center, buccolingually as possible. The entire procedure for the operation and post-operative care is described.

—*M. B.*

597—Goodfriend, S. H.: **The non-surgical treatment of periodontoclasia by means of the pack method.** *D. Items Int.* **62**:36-42. Jan., 1940.

Periodontal infection can be treated successfully by the pack-method; the theory of this method is based on pathological changes of inflammation. There are two fundamental processes in inflammatory reactions —the inflammatory process and the reparative process. The pack method is biologically accurate in its action, since it tends to perform two functions—to destroy infection by bactericidal action, and to aid in tissue repair by its hygroscopic action. Dr. Goodfriend then briefly outlines the three classes of infection—acute, subacute, and chronic.

The pack method is 100% effective in cases of acute inflammation; it is necessary to change a subacute or chronic inflammation to an acute stage, in order that the pack method may be effective. This is accomplished in several ways; cautery by chemicals, electro-cautery, or curetting lining of gingival pockets. The pack is then applied for 24-48 hours at a time, and repeated several times if necessary. The result is gingival recession down to firm healthy tissue. Repair occurs in four days, and is complete in 10-14 days. The simplicity of the pack method enables the general practitioner to treat periodontal disease, as well as the specialist.

—*M. B.*

598—Gottlieb, B.: **The simplifying of pyorrhea treatment.** *D. Items Int.* **62**:538-539. June, 1940.

Every case of pyorrhea can be healed, and the pathologically transformed gingivae can be brought to normalcy. To heal the gingivae, oxygen and paraffin may be used: removal of tartar, and gingivectomy of existing pockets are two further essential measures for treatment.

Pyorrhea cases should be treated daily; at each visit all teeth must be polished, on the lingual and bucco-labial surfaces, with pumice and a soft rubber cup. Removal of tartar is more difficult. To make the field of operation clearly visible, the use of a current (2-3 atmospheres) of oxygen blown into the bottom of the pocket, is advised and transillumination of the tooth, from the side opposite the operative side, with strong light. Use of oxygen makes the field visible, and at the same time the oxygen penetrates the tissues. Use two tubes for spray: one for oxygen, and one to spray hydrogen-peroxide, to remove the blood and the debris while scaling. In this manner, the mild cases can be completed in a week, and difficult ones in not more than two.

—*M. B.*

599—Guillermin, P.: **De l'utilisation des sulfamides en stomatologie.** The use of the sulfamides in stomatology. *Schw. Mschr. Zahnhlk.* **50**:648-650. July, 1940.

The sulfamides must be considered as one of a large class of means for combatting infection; also, it must be understood that they do not replace other proved methods, but must be used in their own place. Dr. Guillermin divides the means of combatting infection into four groups:

1. Antiseptics—which destroy bacteria on the surface.
2. Mechanical means—atomizer, syphon, etc.
3. Biological agents—vaccines, antiviruses, etc.
4. Chemotherapy—specific, non-specific, or specific and polyvalent.

The indication for the use of sulfamide is an acute case. In the choice of the drug, choose the least toxic one, since the life of the patient is rarely in immediate danger. The dosage is fractional and progressively diminishing. Always use, at the same time, local therapy. There is all evidence to show

that the use of sulfamide, in the treatment of septic infections of certain organisms, is as important a revolution as the use of arsenobenzols in the treatment of syphilis; but it must be used with good judgment.

—*M. B.*

600—Haley, P. S.: **Treatment of paradentosis and hypertrophic gingivitis by the use of acetone.** *J. A. D. A.* **27**(4):511-17. 1940.

The author's method is based upon the destructive action of acetone upon living tissue under confinement, and is treatment based upon materia medica. The material used consists of two units: Unit A contains a mixture of pink thermoplastic baseplate material, calamine, and acetone, and cotton twills and balls of suitable size; Unit B contains pink thermoplastic baseplate material softened to the consistency of unvulcanized rubber by the addition of acetone. The cotton balls and twills, thoroughly saturated with the mixture, are packed into the pockets and embrasures; then, a strip of the thermoplastic material of Unit B is placed over the cotton packing, overlapping the gum to seal it. The patient is asked to return in three days, when the packs are removed, and the pockets packed with phenol, left in for twenty-four to forty-eight hours. The process may be repeated; after about three of these acetone-thermoplasticphenol treatments, the pocket tissue will be found largely destroyed. The advantages of this treatment are: no anesthesia; no discomfort; no apparatus need be purchased; no danger of causing toxemia or bacteremia; no tissue laceration; marginal rebuilding eventually takes place.

—*M. B.*

601—Hauer: **Einiges ueber Wipla-Paradentometer, Alveolarnadeln, Kerkhoff-Paste, und andere praktische Hilfsmittel bei der Diagnose und Therapie der Paradentose.** Various aids in the diagnosis and treatment of paradentosis. *Z. Rund.* **49**(11): 369-70. 1940.

A brief discussion of the various instruments and aids in use for the diagnosis and treatment of paradentosis. For diagnosis, both sling and band paradentometers are good; they serve both for measuring the depth of the gingival pockets and as therapeutic instruments. Alveolar needles are small looped needles, which measure pocket depth up to 5 mm. The author himself uses Kerkhoff paste in small amounts with good results, in the medicinal treatment of paradentosis. Treatments should not follow too closely on each other; at each sitting, only 2 or three pockets, at the most, should be treated. Naturally, the patient must practice oral hygiene at home, during the course of the treatment; for this, he recommends 55% alcohol for five days a week, and Paradentosan or Dentiferm for the remaining two days. In cases of severe stomatitis with severely swollen papillae, the symptoms disappear remarkably fast with this treatment; massage of the gingiva is also prescribed.

—*M. B.*

602—Held, A. J.: **Soins paradentaires post-therapeutiques.** Post-operative paradental care. *Schw. Mschr. Zahn.* **2**:169. 1940

Treatment of paradentium creates a relatively unstable state of equilibrium, and this equilibrium must be maintained by keeping a constant check on the constitutional disposition of the paradental tissues. A patient with a disease of the paradentium must remain under constant supervision, and from the beginning of treatment, must be instructed on the nature and development of his condition and the necessity for proper collaboration.

Following a course of treatment for paradentosis or gingivitis, means must be taken to guard against the formation of calculus, circulatory stasis, and occlusal dys-equilibrium; these prophylactic measures are mainly in the hands of the patient. Dr. Held then discusses the various methods for post-operative care, which divide themselves in general, into mechanical and chemical methods. Among the former, mastico-therapy is the most important and is discussed at some length; the various opinions on tooth-brushing are mentioned and the Charter's method recommended as the best. Instruction of the patient in proper tooth-brushing technique must be an integral part of the therapy, and should be checked at subsequent visits.

—*M. B.*

603—Ingersoll, W. B.: **A new and complete treatment of periodontoclasia.** *J. A. D. A.* **27**:1274-1280. Aug., 1940.

It is herein suggested that the chief reason for lack of uniformity of opinions and

methods in the treatment of periodontoclasia is the dearth of fundamental knowledge, possessed by the originators of the various pathologic theories, also a lack of realization of where each theory and procedure applies and where it is deficient.

In the treatment of diseased periodontal areas and in eradication of pockets the author excises all diseased granulation tissues. The author has developed one complete and specific treatment for certain types of cases. It involves:

1. Removal of all upper teeth, if the future of such teeth is in question.
2. Treatment of lower teeth by excision of infected tissue areas.
3. Balancing of the bite relationship.

He defines periodontoclasia as a chronic, non-specific, expanding disease of the alveolar process, causing dissolution of the osseous elements; its clinical manifestations are primarily local, but later are generalized into systemic disturbances. He then reviews and criticizes the various theories as to etiology: uric acid, bacteriologic, occlusion, and nutrition. He himself adheres to what he calls the biologic theory, which considers cell metabolism.

—*M. B.*

604—Jarius: **Paradentose und Paradentose Instrument.** Paradentosis and an instrument for paradentosis. *Z. Rund.* **49**(9):299-305. 1940.

An instrument, designed by the author for curettage and removal of calculus and deposits in paradentotic pockets, is illustrated; he describes the method of using it, and its advantages. He then discusses various other therapeutic methods for paradentosis; the various surgical procedures used by the different operators are mentioned. Articulation and overhanging fillings and crowns should be corrected; missing teeth replaced; radiation treatment with ultra-violet rays is a helpful external therapeutic procedure. Regular massage of the gums by the patient should be enforced; various pastes can be used in addition to the mechanical massage. Internal therapy alone cannot correct paradentosis, since mechanical and exogenous factors are always present. In the course of treatment, the depth of gingival pockets should be checked regularly, to see if, and how much, the treatment is correcting the condition.

—*M. B.*

605—Kallay, J.: **T-Paradentose-Schiene.** T-shaped braces for paradentosis. *Z. Rund.* **2**:48. 1940.

The author has used the same type of brace construction as Dr. Schalow, of which a photograph is included. There is a labial bar much below the gingival margin, and from this, little T-shaped clasps come up to rest around the necks of the teeth; on the lingual, there is also a continuous bar. Through the use of this brace apparatus, it is possible to treat each tooth individually and to fix it lightly in its normal position. In using this apparatus, care must be exercised that no lever arm causes an orthopedic movement. It is especially useful for the upper jaw. Attention must be paid, in all removable bar braces, to articulation and tightness of the brace when applied.

—*M. B.*

606—Kraft, O.: **Ein Beitrag zur Vitamin C-Behandlung der Gingivitiden, Paradentose, und Zahnkaries.** A contribution to the vitamin C treatment of gingivitides, paradentosis, and dental caries. *D. Z. W.* **43**(11):170-71. 1940.

From therapeutic experience, it can be concluded that gingivitis, paradentosis, and dental caries are associated with vitamin C deficiency; it therefore follows that adequate vitamin C administration can help these conditions. Of course, this vitamin deficiency is not the only factor in these diseases, and the other factors must yet be studied and determined. Working with one particular group of people, Dr. Kraft found vitamin C deficiencies ranging from 1100 to 3600 mg., with a resulting average deficiency of 2110 mg.; they came from various environments, but worked in the same place since 1935. A table, shows the incidence of gingivitis, paradentosis, and dental caries in these people. The deficiency was due to the type of food eaten in their place of work, and to the manner in which it was prepared; boiling vegetables and potatoes for long periods reduces the vitamin C content seriously. It is the dentist's duty to ascertain vitamin C deficiencies; and if they are present, the patients should be advised as to the proper foods and methods of preparation.

—*M. B.*

607—Kronfeld, R.: **Clinical pathology of the periodontal pocket.** *J. A. D. A.* **27**(4):499-507. April, 1940.

Periodontal diseases are divided into three fundamental groups:

1. Marginal gingivitis (due mainly to local irritation, but occasionally also to other conditions. 2. Deep, suppurating pockets. 3. Bone atrophy in its various forms.

The second group is the main condition treated by the periodontist.

The author defines normal and deepened gingival crevices, and periodontal pockets. The etiology of periodontal pockets is unknown, but the mechanism of their formation is understood, as well as the tissue pathology. It is formed by a flap of gingival tissue that lies loosely against the tooth surface; the outer wall of this flap is covered by gingival epithelium, while the under wall has a more or less defective epithelial layer, from the inflamed subepithelial tissue of which an exudate of fluid and round cells forms in the pocket as pus. Since the bone atrophy, in cases of suppurating periodontal pockets, may be secondary, the therapy should be directed mainly toward elimination of the pockets and control of suppuration. There are two main groups of therapists: those who advocate removal of the entire flap of tissue forming the pocket, and those who believe that a reattachment of the gingival tissues to the root surface is possible. As for prognosis practically all that the dentist can do is to prolong the period of usefulness of the teeth.

—*M. B.*

608—Leriche, E.: **L'éther carbonique neutre de quinine comme adjuvant au traitement des stomatites.** Neutral carbonic ether of quinine as an aid in the treatment of the stomatitides. *La Rev. Odont.* **62**:82. April, 1940.

Like most authors, Dr. Leriche recognizes local and general causes; but,—and this is the idea he presents and defends—each specialist must be able, before any course of treatment, to relieve his patient. For this, he should have at hand a medicament which will answer this need; such a drug is neutral carbonic ether of quinine (quinenol). The arsenobenzenes are effective only against the fuso-spirilla, and vaccines act too slowly, so these cannot be used.

After giving the history of quinine as used in stomatology and indicating its chemical and therapeutic properties, the author presents some personal observations of cases of stomatitis (medicamentosa, following eruption of lower third molars, in pregnancy, in pyorrhea, and in hypertrophic gingivitis) in which he obtained excellent results with this drug. Thanks to this drug, he has secured rapid elimination of pus and pain, allowing him to better apply appropriate therapy, secondarily, in each case.

—*M. B.*

609—Lundquist, G. R.: **The pathology and treatment of so-called pyorrhea.** *J. of Tenn. D. A.* **20**(1):10-15. May, 1940.

This presents nothing essentially new but merits reading because of the broad scientific treatment of the subject as a biologic problem. Author emphasizes the fundamental importance of the deep gingival sulcus as a factor in producing and maintaining the disease process. The relation of stress injuries to the successive inflammatory changes is admirably explained. Success in these, as with other chronic diseases, is largely dependent on whether treatment is instituted when the disease is on the rise or on the recovery, or fall side but often even in cases of failure at the outset, correct treatment if continued until the high reactive point is passed will lead to cure. A general outline of treatment is given. Electro-coagulation should not be attempted until all possible deposits have been removed from root surfaces; and then it often follows that the radical removal of the soft tissue side of the pocket will be found to be unnecessary.

—*C. R.*

610—Lundquist, G. R.: **The zero crevice in the management of periodontal involvements.** *J. A. D. A.* **27**(6):837-50. 1940.

In the protection of the skin or mucosa against infection, the mechanical integrity of the skin or mucosa is of the utmost importance, but the resistance of the underlying tissue to penetration by bacteria is equally important. Gingival crevices seldom have an intact epithelial cover; also, the connective tissue under the epithelium does not offer much protection against further invasion by bacteria once they have penetrated through the epithelium. Therefore, the elimination of the gingival crevice or the production of an intact epithelial lining on the soft tissue side of the crevice, with a sound

underlying connective tissue base, is desirable. Attention must be called to the fact that the soft tissue normally changes its position from the crown of the tooth to the root of the tooth with age; also, the tooth tends to change its position in more than one direction.

The various causes of gingivitis are discussed. Correcting diet deficiencies and eliminating metallic poisons, if present, will aid in a hygienic approach to the problem of pocket elimination, as far as shallow crevices or pockets are concerned; but removal of deep pockets is another problem. Removal of concretions on the hard tissue side of the pockets is necessary, and also correction of any existing traumatic occlusion. For the elimination of the soft tissue side of the periodontal pocket, either electrocoagulation or surgery can be used. The aim in all cases is to achieve a zero, or near zero, crevice with a natural contour for deflecting food particles, to avoid further injury.

—M. B.

611—Lyons, H.: **Drugs in periodontal therapy.** *J. A. D. A.* **27**(6):879-86. 1940.

The various types of drugs used in the therapy of periodontal diseases are reviewed, in an attempt to debunk some of the fallacious claims made by manufacturers and advertisers. In mouthwashes, we can conclude that a warm, slightly hypertonic solution of sodium chloride has all the good features of any mouthwash and is devoid of all its potential dangers; solutions having astringent properties tend to restrict circulation and so are not to be recommended. Dentifrices can act only as an adjunct to the mechanical cleansing action of the toothbrush, and in no other way; it is the dentist's duty to enlighten the patient in this regard. Pyorrhea astringents, on the whole, are harmful, and at best are useless; they tend to form irritating coats of proteinates over the mucosa, and, more important, cause vasoconstriction which delays healing processes. Antiseptics and germicides have also been grossly overrated. First of all, the bacterial factor in most periodontal lesions is non-specific; secondly, even in acute Vincent's infection, in which their use has been most advocated, the acute inflammatory condition tends to reach its peak and then die down even without medicinal interference; and thirdly, the action of an antiseptic lasts only a very short time, and then the oral mucosa is again covered by bacterial colonies.

Pocket dressings are of little value, and may actually delay healing; a normal blood clot is the best dressing. Epithelial solvents are of no value at all, and may be extremely harmful; caustics, also, are often very harmful, and remove granulation that represents an attempt at healing. For pre-operative treatment, there are many drugs to quiet the patient and remove much of the fear and discomfort of periodontal therapy; there is also a wide variety of drugs for post-operative care. The article closes with a plea to dentists to use only drugs listed in *Accepted Dental Remedies*, which are of established value and of safe therapeutic action.

—M. B.

612—Pelzer, Rudolph H.: **A study of the local oral effect of diet on the periodontal tissues and the gingival capillary structure.** *J. A. D. A.* **27**(1):13-24. Jan., 1940.

The author investigated oral and gingival changes following two types of diets, one strongly detergent, attrition-producing; the other predominantly non-detergent, soft food. Direct microscopic examination of the gingival capillaries was instituted, and the process is described. Normally these vessels in vivo tend to maintain their "posture or tonus" at around 6 to 8 microns diameter and these figures are taken as a basis for comparison and for correlation with clinical findings. The individuals on a hard detergent diet showed a characteristic oral syndrome of "(a) wear of the morsal and proximal surfaces of the teeth; (b) a well-developed facial masculature; (c) the presence of small medullary spaces and dense trabeculation of the maxillary and mandibular bones; (d) little tendency to calculary deposition; (e) the presence of well-hornified, resistant gingival tissues; (f) a low caries index, and (g) according to gingival capillary microscopy, normality of gingival capillaries in number, distribution, and tonus." Those using soft non-detergent diets showed an equally characteristic syndrome

but exactly the reverse of the other. The clinical and pathological findings substantiated the capillary findings "evidencing the basic validity of gingival capillaroscopy."

—*C. R.*

613—Prosser, T. E.: **Comments on periodontal disease.** *D. Digest.* **46**:91-94. Mar., 1940.

The gingival crevice or pocket is formed by the surface of the tooth on one side and the free margin of the gums on the other; this free soft tissue is covered by stratified squamous epithelium. The depth of this crevice normally varies with age.

Scaling, polishing, and the application of drugs can produce excellent results in many of the simple forms of periodontal disease; when the gingival pocket becomes deep, however, surgery is imperative. Pre-operative sedative is given, and infiltration anesthesia is used; then, the gingivectomy is performed with the proper instruments. After the tissue is fully removed, pledgets of cotton soaked in tincture of metaphen are forced into the curetted spaces; they are removed, after a few minutes, and a surgical dressing applied. Dr. Prosser finds that sulfanilamide powder added to the dressing hastens healing. The dressing is changed the third day after the operation, and then every 4-5 days for at least two weeks. When recovery is sufficient to remove the dressing, the patient is given a pair of hemostats and small cotton rolls, and instructed in massage of the gums.

Concerning the newer coagulation method, Dr. Prosser considers that the resulting coagulum, or mass of decomposing tissue, is not a desirable thing near a wound, and its presence delays healing.

—*M. B.*

614—Sabeff, P.: **Kleinsche Untersuchengen ueber die Anwendungsmoegluchkeiten der Kaffeekohle in der konservierenden Zahn heilkunde.** Clinical studies in the application of coffee charcoal in conservative dentistry. *Z. Stomat.* **38**:134-144. Feb., 1940.

Coffee charcoal, which is today rather too noisily acclaimed from various sources, is useful in:

1. Absorbing and mechanical cleansing, like every medicinal charcoal.
2. Phenols found in roasted coffee have disinfecting action; they would also increase action of sulfo-cyanate ion in saliva.
3. Through greater capillarity and small amount of tannic acid, it renders the environment unsuitable for bacteria.
4. Caffeine produces active hyperemia.
5. Vitamins and "growth substances" should also be present.

Fresh preparation is best; it should not be over-roasted, but brown and still smelling of coffee. The best action was found by the author in gingivitis, stomatitis ulcerosa, gingival bleeding, "foeter ex ore." In paradentosis, those cases with acute inflammatory symptoms of gums reacted well. Through the use of coffee charcoal medication, further bone atrophy can be prevented and the disease process can be brought to a standstill.

—*M. B.*

615—Smith, R.: **The systemic aspect of periodontal disease.** *D. Digest.* **46**:378-382. Nov., 1940.

The cases discussed are comparatively young. Medical examinations showed that the most common abnormalities associated with diffuse atrophy are secondary anemia (hypochromic microcytic type) and the lack of normal thyroid function; some of these patients, however, were normal in these respects. Proper medical treatment was given, after detailed medical examinations, but with this alone, none of the cases showed any improvement in dental condition. Local periodontal treatment, together with restorative dentistry, when indicated, proved to be most effective.

Periodontal treatment consisted of prophylaxis, subgingival curettage, and flap operation of gingivectomy. Dental treatment consisted of: relief of occlusal trauma, and restoration of dentition to as near normal function as possible. It seems impossible, in many cases, to arrest completely the destruction of periodontal tissue, once begun; but periodontal treatment, with medical assistance when indicated, will usually control bone resorption, delay loss of teeth, and prevent involvement of remaining unaffected teeth. However, alveolar diffuse atrophy is still a problem awaiting intelligent solution, based on greater knowledge of etiology and prognosis.

—*M. B.*